The Journal of
Laryngology and Otology

(*Founded in* 1887 *by* MORELL MACKENZIE *and* NORRIS WOLFENDEN)

November 1961

CONCERNING THE PATHOGENESIS OF DESTRUCTIVE EAR DISEASE

By F. McGUCKIN (Newcastle-upon-Tyne)

THIS paper is based on the material used for the Annual Address given at the Institute of Laryngology and Otology on November 25th, 1960. Dr. Louis Verhoeven and I had hoped that the James Yearsley Lecture of 1961 and the present work would be published jointly and that we might find it possible to include nearly a hundred colour illustrations. Unfortunately technical and financial difficulties have proved insuperable and we have been forced to abandon the ambitious project of illustrations despite the more than generous help provided by the University of Durham Photographic Department.

It is my purpose to consider the disease which for many years has been called chronic otitis media and to decide whether or not our traditional concept is any longer tenable.

I would begin by describing three cases shown to me by Verhoeven in Antwerp and I use them firstly because his photographic work is of first rank and therefore the records are there for all to see, and secondly because he has become almost as eccentric as myself about the subject to be discussed. (Eight colour slides followed.)

We have together studied the ears of a highly intelligent young man holding a responsible post in Belgium. His left ear shows a physiology lying somewhere between the mechanism of a Wullstein type 3 and type 4, and it provides an average efficiency of 20-25 db. below the European zero line. Neither the patient nor his parents, who are also intelligent

people, are aware that there ever was any trouble with this "normal" ear, but for many years they have been very much aware of disease in the opposite ear which had already been submitted to two operations before being seen by Verhoeven.

The second patient, a young man of about 19, came because he had learned that an uncle was going deaf, and he wished to know whether he himself might go deaf in the future. He was unaware of ear trouble at any time and considered the hearing normal in each side. Nevertheless the right tympanum showed extensive old destruction, with the stapes isolated above a thin round window baffle which could be followed round the hypotympanum to the tubal exit. The "regenerated" membrane was so thin that all the details of the hypotympanum and the tubal exit could be seen through it. One can only conclude that the patient grew up with unequal hearing in the two sides, though with binaural social hearing, and since he never knew anything different, this to him was normal.

The third patient was a young lady of 19 who presented with a fœtid left otorrhœa and some deafness. The discharge had developed without pain and was smelly from the start. Under local anæsthesia and using the Zeiss microscope, masses of pus and sodden keratin were removed from the canal by suction. After meticulous cleaning the eroded posterior attic was seen to be filled with a mass of keratin (or cholesteatoma, or epidermosis) which invaded down behind the ear-drum into the mesotympanum. The attic was cleared by suction, the postero-superior segment of the drum-head opened and cleared in similar fashion, and the inner-wall mucosa noted to be in good condition. Photographs taken 2 weeks later suggested that this simple suction-clearance performed through a speculum had controlled the disease and restored useful hearing. I saw the lady ten months later and all was well.

In these examples, a few among many, there was ample evidence of extensive tympanic destruction and of healing which was wholly spontaneous or assisted only in a small way. I was brought up to believe, or at least allowed to assume, that this kind of picture represented the fortunate end-result of spontaneous termination of chronic otitis media. I do not, nowadays, accept any such explanation, and for the moment the position may be left there in suspense.

In September, 1960, I was asked to see a little Canadian girl 8 years old. For 4½ years the left ear had produced a continuous foul discharge, uninfluenced by cortical mastoidectomy and several upper respiratory operations. After taking a detailed history, the good hearing in this left ear caused me no surprise. (Audiogram shown.) By all the standards of traditional teaching this was a long-standing cholesteatomatous chronic otitis media, but after two suction-clearances we knew that the mucosal element of the drum-head was still intact, and consequently that there was hope. Chronic otitis media has been my professional hobby for 31 years

and now I come not to praise the disease, perhaps not yet to bury it, but certainly I come to measure the body.

Nowhere in the literature is there a strict definition of the disease, and to my shame I have for years evaded the issue by using description. I would now confront the definitive problem.

We may perhaps agree that the word chronic has something to do with time, and presumably a chronic disease must be one running a course significant in the lifetime of man. For me, it also implies a suggestion of irreversible damage, always physical and sometimes functional. The term otitis means inflammation related to the ear, and when we add the word media, we mean, or should mean, an inflammation of the middle-ear cleft, something which belongs to the cleft proper.

Supposing for the moment that we accept this concept as meaningful and not mere playing with words; then it seems to me that chronic otitis media in my part of the world is becoming a comparatively rare disease, though we still see an immense mass of destructive ear pathology. Moreover I now believe that the disease as already defined was always less common than we thought, even in the days of severe acute middle-ear suppurations which were so damaging as to be destined to become chronic because of nutritional injury.

Cholesteatoma, or hyperkeratosis, or epidermosis, is not in itself an inflammatory lesion, though inflammation may be added; it always begins outside the middle-ear cleft and, when it invades the cavity, any otitis that may supervene is purely incidental and secondary. Dissolution of bone by clean cholesteatoma, or by dry keratosis, or if you like by epidermosis—and I do not see any fundamental difference between their actions or even their genesis—such dissolution of bone is achieved neither by infective inflammatory change nor by pressure, but by some as yet ill-understood chemical and possibly enzymal process. These noxious conditions, so far, are not inflammatory and they do not necessarily belong to the middle ear. They are therefore not chronic otitis media. Since I hold that the great bulk of so-called chronic otitis media arises by cholesteatomatous, or keratotic invasion from outside the cleft and not in consequence of disease initiated within the cleft, then I must logically conclude that by definition chronic otitis media is relatively uncommon.

Last January, Mr. Brian Pickard (1960) published three cases of necrosis of the tympanic ring. At operation he had been surprised to see that in each case a long-standing otorrhœa was consistent with strictly limited disease, since surgical exposure of the cleft had revealed no pathology except in the region of the posterior marginal perforation.

On November 3rd, 1944, Mr. Mollison (1944) joined in the Section discussion, speaking of mishaps. I quote his words "(*a*) Opening the mastoid under the impression that there is a posterior marginal perforation

with granulations and pus, only to find the mastoid normal and granulations due to local necrosis of the tympanic ring, is always annoying". In the same discussion Mr. Layton (1944) said "We had all learnt that chronic ear disease was an incurable condition. Ear disease had to be tackled from an earlier stage than at present. In the population it was to-day dealt with from the schools. That was too late. It had to be dealt with before the child got back to school after the acute ear disease had occurred". Mollison made an observation which was entirely right. Layton expressed an opinion having much of truth in it, but it was in part based on the false premise that chronic ear disease must be a sequel to an obvious acute incident.

Now the interesting thing about Pickard's three cases is that, so far as is known, the patients never had an overt acute episode. The histories were competently taken but their significance was overlooked. (I was able to show Mr. Pickard or Dr. Lindsay Gray the case of a man having a continuous history of 45 years of fœtid otorrhœa cured by one out-patient treatment.) As far back as 1874 Von Tröltsch (1874) allowed that perhaps some cases of chronic otitis media might be consequent on lesions beginning in the external auditory canal, a notion given some support by Albert Gray (1910) in 1910. The general idea is, therefore, not altogether novel. Whilst talking of our distinguished predecessors I read somewhere, perhaps it was in Politzer, that no one had ever observed the formation of posterior marginal perforations. I have observed such formation and I think it arises by invasion from the external canal and not in consequence of disease beginning inside the cleft.

If the histories of all patients suffering from chronic or relapsing otorrhœa are elicited with loving care then it will be seen that the text-book teaching of the acute-chronic sequence is to-day almost wholly invalid. I repeat that destructive ear disease is still common, but I also hold firmly that most of it does not justify the term chronic otitis media. Furthermore, the observation is of prime importance if we are to possess another preventive weapon in the war against chronic aural damage.

I became interested in keratosis of the external ear in 1931, but from my operative notes it would seem that 16 years elapsed before I began to see what my eyes beheld; and it took another ten years before I dared to confess even to myself that almost all of our traditional teaching on the genesis of chronic otitis media was misleading. And it was only in October, 1959, in Kansas City, that I first summoned the courage to discuss my views in public, and to prepare for the interment of the disease. Eleven years ago, at a Section Meeting, I mentioned attic lesions which appeared not to suppurate at all but which nevertheless caused attic damage (McGuckin, 1950). Eight years ago, also at a Section discussion, I remarked that we saw an increasing proportion of patients in whom aural

disease was already chronic when the first symptom was experienced (McGuckin, 1953).

Almost against my will, I have been compelled to accept the evidence offered in such great wealth by innumerable patients with ear disease. The concept to be put forward may be startling and it does mean that I must eat many of my own words, including most of what was said as recently as the Yearsley Lecture of 1958.

Lest you complain that here again we have the same verbal gymnastics as ever, I will submit a tiny fraction of the evidence, a few pictures derived from hundreds of patients. In the process you may find the wheel turned full-circle; you may find yourselves in the post-tympanoplastic era and to some extent back in the world of Tumarkin's atticotomy, with I hope some idea of how Tumarkin achieved what he did. True chronic otitis media generated from damaging or widespread acute pathology inside the middle-ear cleft has been all but eliminated by the advance in medical therapy, at least for the time being. Why then are we left with so much destructive ear disease? The answer surely is that there always was in the mass of pathology loosely called chronic otitis media a hard core of material which never belonged primarily to the middle-ear cleft and herein lies the error of blind acceptance of disease labels. I had for years been trying to define a disease which hardly existed, and it is no wonder that the task proved difficult.

(At this point 49 colour slides were shown in groups illustrating various stages of operative microscopic investigation and the facts arising therefrom, including the following: massive keratotic destruction of outer, middle and inner-ear leaving only the facial nerve; cases of gross exposure of facial nerve consequent on destruction of the bony floor of the external auditory canal; cases showing loss of bony canal below the tympanic annulus so that the membrane appeared to be suspended in mid-air above, and also the mechanism of formation of a "false fundus" external to the tympanum; examples of symptomless erosive keratosis discovered at routine examination of the so-called good ear; one case in which it was known that a considerable mass of keratin had accumulated in 8 days; a case of bilateral keratosis first presenting in a lady of 67 who complained only about the left ear which showed a grossly granulating lesion at the inner end of the meatal fibro-cartilage; numerous examples of extremely thin "regenerated" membranes completely lacking a middle layer in the drum-head; one patient in which the actual formation of a posterior marginal perforation could be demonstrated; several cases showing keratin inside the sinus tympani and a further number showing spontaneous regeneration of simple round window baffles; one group of slides was used to demonstrate that keratotic invasion of the attic could pass inwards to arrive inside the labyrinth, or downwards deep to the drum-head, or backwards through the aditus, and finally backwards to produce

very wide exposure of a perfectly normal blue lateral sinus; some of the pictures were used to indicate that inflammation in any ordinary sense was quite unnecessary to gross destruction.)

Erosions Caused by Hyperkeratosis

Certain points which arise from the pictorial evidence just reviewed should be discussed here. Hyperkeratotic lesions may at times be first observed as small sticky crusts over the deep bony wall of the external canal. If the crust is lifted, sodden keratin is revealed and when it is removed there may be a little fine granulation tissue overlying a shallow osteitic lesion in the bone. This process may move towards the region of the annulus. If at any given moment there is a wide attack on the nutrition transmitted across the posterior annulus, then the middle element, and the mucosal layer of the tense membrane are destroyed, and so a perforation is formed. However, if at any one moment the attack is localized to a small fraction of the ring and if there is progressive spontaneous arrest, then we see the gossamer membranes perhaps attached inside the annulus and even to the pyramid itself. We are all familiar with these fine membranes and we know that in certain circumstances the appearance is consistent with hearing which is as near normal as makes no matter, and we also know that very often there is no history of ear disease. This matter cannot be dismissed by thinking that patients and their parents are all unintelligent people. In medicine as a whole we are accustomed to lean very heavily on the evidence offered by the patient and there is no reason to dismiss this evidence when given by a person suffering from aural disease.

Associated Lesions

Much has been made of the triad consisting of bilateral so-called wax keratosis, chronic multi-sinusitis and poor bronchial epithelium perhaps amounting to bronchiectasis. In my experience these associated lesions belong only to the minority of cases showing hyperkeratosis in the ear. Moreover the mere contemplation of the triad cannot be expected to cure or alleviate the ear complaint. It is reasonable to suppose that a person suffering from sinusitis or bronchiectasis would be rather better off if his ear troubles can be alleviated and the hearing improved.

Again, the association of secretory otitic fluid and keratosis has often enough been demonstrated. Secretory fluid is seen in the minority of the cases, and the fluid may be strictly regional, perhaps limited to a small sub-section of the mesotympanum, to one or other of the attic spaces or even to part of the aditus. I have no hesitation in removing such fluid if it presents as a complication.

"False Fundus"

The study of this material has convinced me of the manner in which a false fundus may be formed in the external auditory canal approximately one centimetre or so lateral to the level of the annulus. An erosive keratotic disease may attack the inner edge of the fibro-cartilage. When the attack is limited to a small segment, a mass of granulations is observed, or simply a swollen fibro-cartilage possibly giving rise to severe pain. When a granulating lesion extends all round the clock it may eventually "heal" together by a fibrous diaphragm of saucer-shape, looking at first sight like a rather curious drum-head too close to the observer's eye. Beyond a web of this kind there may be no active disease, though the ear is necessarily partly deafened; on the other hand erosive disease may be enclosed beyond the diaphragm and the case may present with an acute exacerbation, or perhaps even with a complication such as an intracranial spread, a labyrinthine erosion or a facial paralysis.

Other Perforations

I was taught to believe that a simple perforation was commonly the result of destruction arising from pathology inside the middle-ear cleft. An extensive experience of microscopic investigation suggests that this view is not correct. If a granulating intrinsic myringitis is observed very closely one will almost invariably see small masses of sodden keratin in between the lobulated tissue and even deep to it. It is possible in many cases to remove this granulation tissue with tiny suckers and leave the underlying mucous membrane of the drum-head intact. More advanced lesions will show that not only the middle element but also the mucous membrane have been destroyed, so that there is a true gap in the membrana tensa. The finding is not inconsistent with a normal mucosa on the promontory. My suspicion is that a central perforation arises far more often in this fashion than from disease which penetrates from within outwards. Perhaps one may go further and say that the same mechanism may apply to the extreme marginal perforation exposing the tubal exit.

Natural Healing and Its Implications

The recognition of natural healing first caused me to consider the possibilities of reconstructive surgery. Years later, and after much heart-burning, the same kind of observation started for me a gradual retreat from the standard methods of tympanoplasty, something which has rapidly developed into a major retreat. Many of my notes read as follows, as in the case of Mrs. M. C. Right endaural excisive operation leaving forlorn type 4 conditions. After such an operation in Mrs. M. C., a membrane gradually regenerated to form a roof half way down the mesotympanum.

The result was a rather thick baffle but a natural one over the round window providing good communication with the eustachian tube. Above this level there was another regenerated membrane attached either to the stapes or to the pyramid. In some sense this approach may be regarded as one variant of the modern tympanoplasty, consisting chiefly of the preservation of what may be a forlorn hope and then seeing what nature can do. The result in the case mentioned is that the ear has very useful hearing. I cannot work out whether the physiology arises from a type $3\frac{1}{2}$ or a type $4\frac{1}{4}$, but it does work (slides shown).

Many of us have for so long been obsessed by what Scott Stevenson condemned as the lure of technique, many of us have become so lost in the labyrinths of otosclerotic and dry temporal bone surgery, that we have quite forgotten, or perhaps never knew, that nature had her own little tricks which might require no assistance or just a little assistance. It is an absorbing business to try to work out all permutations and combinations which are possible in the variety of physiological systems consistent with useful hearing. It may be convenient, it is convenient, to adopt the five types of Wullstein, but I think that sometimes we may have a type $1\frac{1}{2}$ or $2\frac{1}{2}$ or $4\frac{1}{4}$ and still have useful function. We may see damage repaired so well as to approximate to normality or type 1, or we may see the reverse type 4 described by Garcia-Ibanez, or a varied collection of many types. Nature is quite wonderful if we will only give her our attention.

It is of immense importance to note how often there is some kind of useful physiological structure remaining beneath a suppurating destructive mass of keratosis; and this applies quite frequently when invasion has become widespread in the middle-ear cleft. It is a common experience nowadays that what nature leaves behind is almost invariably more serviceable than anything the surgeon can reproduce. Moreover, when operative intervention is followed by spontaneous regeneration of no more than a good round window baffle, the hearing is nearly always socially adequate and it may be above the 20 decibel level.

Use of Skin in Reconstructive Surgery

If skin indigenous to the ear itself so often becomes unstable and even offensive, it is not surprising that there has been widespread trouble from some of our more recent efforts. I have considered how far the implantation of skin may be useful in reconstructive surgery and I am gradually coming to think that the best physical and functional results have occurred in those cases where skin grafts have merely acted as a scaffold for regeneration before their complete rejection and ejection. The idea first crossed my mind in the middle of a lecture when with some pride I was showing a slide of a highly successful type 3 reconstruction. The following slide represented a natural regeneration of, or preservation of an extremely thin membrane and

it struck me that the appearance in each was almost identical. There was certainly no implanted skin in the second case and it seemed probable that in the first case the skin used for reconstruction had been thrown out in the washing. The more these cases are studied microscopically the more convincing becomes the hypothesis (photographs were shown).

There can be few of us, with any experience of tympanoplasty, who have not been troubled by the post-operative behaviour of *extrameatal free skin*, whether the graft be of whole skin from behind the ear or split skin from elsewhere. Even in the immediate region of the mesotympanum the graft often enough produces a desquamative mess, as one might perhaps expect from differences in structure, in cellular reproductive capacity, habitat, temperature and so on. When it does survive, the graft may require constant attention because of desquamation and in this respect the position is in strong contrast with what can be seen to occur when mucosal regeneration takes place before rejection of the graft. When skin is placed beyond the limits of the mesotympanum, thereby reaching areas with much poorer blood supply, its behaviour is notoriously bad. Moreover both here and in the region of the mesotympanum, the behaviour may be good for 6 months, a year or longer and thereafter it may break down in the mastoid to form a granulating mess, or it may perforate over the mesotympanum—in which event the new drum-head has gone!

If these statements seem rather forthright then I would ask you to consider why such a variety of methods has been employed to replace the use of extrameatal free skin. In one clinic, meatal skin only is used when available, and it is by no means always available (Plester, Dusseldorf); in a second, pedicle skin flaps are employed (for example by Beales, Doncaster); in a third clinic, long bone periosteum is used (Bocca, Milan); in a fourth, pedicle grafts of temporalis muscle are fashioned (Rambo, New York); in a fifth, sheets of very thin polyethylene have been tried, presumably in the hope of encouraging mucosal regeneration (House, Los Angeles); in a sixth, vein grafts are employed (Shea, Memphis); and my own tendency for the present is to hope for regeneration possibly in association with thin sheets of nylon. We need to consider what may be the best method to prevent enclosure of, or ingrowth of keratinizing squamous epithelium.

There is one common denominator in all of this, namely that there is widespread dissatisfaction with the implantation of extrameatal free skin, a dissatisfaction which to my knowledge extends right across from California into the continent of Europe. My own method has tended towards the abolition of the standard tympanoplasty as far as possible, and I have come to depend very much more on preservation of tissue and the hope of regeneration. Early on I employed large sheets of post-aural skin and later reduced these to mesotympanic and attic grafts only.

The next stage was the use of split skin for local reconstruction and to supplement this by another split graft over the posterior soft tissue edge of the endaural wound. The latter graft was then abandoned and even the mesotympanic graft has almost wholly given way to the expectation of regeneration.

There was also a general move in another direction, towards the meticulous microscopic investigation of every case presenting the picture of what I had long called chronic otitis media. It is true that repeated minor interventions commonly consisting of microscopic suction-clearance have often been necessary to control disease and that sometimes it has been necessary to fall back on major exploration of the middle-ear cleft because control was not effected. It has, however, been most surprising to discover how little intrinsic chronic middle-ear disease there is, and equally surprising as well as gratifying how many cases have been brought under control with preservation of good function. Furthermore, minor interventions have on occasion been extended to such procedures as atticostomy, ossiculectomy, aditostomy and so on, still working down a speculum.

A remark made by Thorburn in the Yearsley Lecture of March, 1960, may be recalled here. He suggested that the time might not be far off when every otologist should investigate all ear lesions under the microscope. This has been the habit of Louis Verhoeven for two or three years now, and it is also my habit—with a difference, in that I do far more of my cases under general anæsthesia than he does. More recently I have extended the practice of these investigations so that a much larger proportion of patients are dealt with without any anæsthesia at all.

Whither?

It may be well to look back over what has been said and, assuming that there has been anything in it, to decide where it all leads. I suggest that we are arriving in what might be called the post-tympanoplastic era but in so doing I have no intention of speaking disrespectfully of the men who made the initial accidental observations of the various physiological systems consistent with useful hearing, or of the brilliant work by which they attempted to reproduce these systems surgically. We are in our present position because of this kind of work, because we have learned to see what our eyes have been looking at for so long, assisted of course by the enormous improvement in optical equipment during the past few years. It also looks as though we have arrived in an era when true chronic otitis media is a comparatively rare disease, always allowing that we pay proper attention to the meaning of words. This could be an important step since it may be prophylactically significant.

Many years ago I was troubled by a clinical problem, namely the

occurrence in small babies of thick ear discharge which was fœtid from the beginning, sometimes without evidence of obvious pain. I suggested to the late Sir James Spence the limited ambition of finding someone competent to observe accurately all the ears of babies and children admitted to two large hospitals. This ambition, limited though it was, was never achieved, but I am sure that it is the kind of investigation which would solve the problem of destructive ear disease in young people.

Pathogenesis

I am not competent to enter into a discussion about the possible causation of keratotic activity in the ear. My colleagues in dermatology and in pathology tell me that the process can only be described as a hyperkeratosis and that the liberated squames contain no nuclei whatsoever. So far, my investigations have got nowhere and I do not think that my training in the experimental discipline is good enough to set up the right kind of enquiry. I know very little indeed about the essential structure of deep meatal skin or of the cuticle of the tympanic membrane, and still less about the rate of reproduction of surface cells, except to say that it is clearly very much less than that of the remainder of the skin of the body. Furthermore I have no knowledge of either the direction of or the speed of migration of surface cells from within outwards. I cannot even say whether an error in normal migration is a possible factor in the pathology which may arise from accumulation of pure keratin in the external auditory canal. I can speculate on some factors which may accelerate a process or a tendency already present. Perhaps I am reasonably sure that given unstable conditions, the entry of water and particularly of ear-drops may convert a lesion which has been harmless into one which becomes offensive. It also seems possible that pyrexia from any cause may change a dry mass of keratosis into a wet suppurating mass, whether it be in the child or the adult.

There is a vast field of inquiry still to be explored in the disease we are considering. The first need is to accumulate facts and then a willingness to accept the concept that the disease is not only potentially destructive but that it is common. We need to record mentally the findings which arise from routine observation of patients who have no complaint about the ears, and more particularly the most minute variations from the normal which may exist in one ear when the patient complains about the other ear. If we can gather a corpus of well-established clinical data, then we must find the men who have the skill and the talent to formulate the right kind of experimental enquiry. It may be that in all of this there is a need to jettison the great bulk of what we have been taught to believe about destructive ear disease. If this is a revolution which at first sight appears to be unacceptable, then I would only ask you *not* to reject the

thesis out of hand. Looking back over the past 30 years, it has been my experience that many revolutionary concepts have been ruthlessly condemned in the beginning and some of you will remember the prejudice with which the work of Lempert was initially received. You may also recall the lively controversy about the new idea of the medical treatment of acute suppurations in the middle-ear cleft and the disparaging remarks about verbal gymnastics when novel ideas were put forward in terms of accessibility and inaccessibility, reversibility and irreversibility, excisive surgery as opposed to radical surgery, and so on. Sooner or later, and it is important to hope that it will be sooner, this revolutionary approach to destructive ear pathology will be accepted, though in the process it may be largely modified. I only ask that the evidence given by a multitude of patients and the evidence offered to the eye which is willing to see, be not ignored.

The regenerated membrane. The term regenerated membrane has been used frequently in this lecture. It is certain that sometimes repair is effected by regeneration and some slides have been offered in confirmation. On the other hand the word regeneration cannot always apply, since the destructive process may be arrested either by nature or the surgeon, before there is a true penetration inside the middle-ear cleft proper. In this event preservation of the membrane might be a more correct description than regeneration.

However, it may be helpful to consider what we already know of the visible process by which regeneration is effected. It is not possible with present photographic equipment to show all that the human eye can see under magnifications of 16, 25, and occasionally of 40. The eye can see that even in very old perforations with densely fibrosed edges, the outer cuticle of the membrane pushes out the thinnest possible tendrils of keratin flakes which, when fibrosis is gross, curl up and turn outwards before reaching the very edge of the gap. Where fibrosis is virtually non-existent these tiny flakes may cover the gap if it is small enough—and if the effort is widespread enough. The result is not successful unless mucosa, even of the thinnest possible kind, grows beneath the fine cuticle. How do I know that this is so—only by the most scrupulous observation under high magnification, because in this event the trained eye is able to see that successful closure of a gap which contains no middle layer, depends on a layer of tissue containing recognizable vessels. When there is only cuticle no vessels can be recognized under the highest magnification we have available. When there is a mucous membrane beneath, these vessels can be demonstrated but not as yet pictorially (two slides were shown in an effort to illustrate this point).

Here we have a sphere of observation in which the working clinician must play the leading rôle, and in which he must pose his problems to an understanding otopathologist, to a person with nearly as much clinical

training as the surgeon himself and very much more knowledge of laboratory techniques.

The whole field of medicine offers nothing more fascinating than the vision of nature at work, fashioning her own tympanoplasty. We may watch the reconstruction day by day, or week by week, or month by month, according to the speed of repair. Nevertheless it is far far better to detect and to arrest pathology when we can, so that nature may preserve a normal physiological system or something very near to it.

I have seen keratosis in a child of 3 months, and one of many exacerbations in a lady of 86. I have seen the disease present as something active for the first time at the age of 76. In our clinic we are able to produce cases by the score for the delectation of visitors. We have any given number of demonstrations. Please do not dismiss the concept as the crazy notion of one who is almost as unstable as the unstable cuticle he has been describing.

REFERENCES

GRAY, A. (1910) "The Ear and its Diseases". London, p. 182.
LAYTON, T. B. (1945) *Proc. roy. Soc. Med.*, **38**, 419.
MCGUCKIN, F. (1950) *J. Laryng.*, **64**, 693.
——— (1953) *Proc. roy. Soc. Med.*, **46**, 371.
MOLLISON, W. M. (1945) *Proc. roy Soc. Med.*, **38**, 418.
PICKARD, B. (1960) *J. Laryng.*, **74**, 59.
TRÖLTSCH, A. F. VON (1874) "The Surgical Diseases of the Ear". London, p. 62.

Elmbank,
Clayton Road,
Newcastle-upon-Tyne, 2

THE SEVENTH JAMES YEARSLEY LECTURE

THE EXAMINATION OF THE EAR IN CHRONIC OTITIS MEDIA*

By LOUIS VERHOEVEN (Antwerp)

CHRONIC otitis media has been and still remains a major problem for the otologist. Every case is different and in each an accurate diagnosis must be made so that proper treatment can be undertaken.

Whereas it is easy to diagnose otosclerosis, it is very difficult to make an exact diagnosis and subsequently to choose the most suitable method of treating the complex problems presented by chronic otitis.

After Wullstein and Zöllner had suggested in 1953 that the treatment of choice in chronic otitis should include an attempt to reconstruct middle-ear function by means of a tympanoplasty, many otologists throughout the world believed that major surgery was indicated in all cases. However, a careful appraisal of this view made after studying patients has proved that this statement is no longer tenable. The progress of audiology, the use of the binocular loupe, and most of all the introduction of the Zeiss oto-microscope have not only made aural microsurgery possible but they have also given the otologist the opportunity to make a much more careful examination of the middle ear in chronic otitis media.

Chronic otitis media can be divided into two distinct types. Firstly, there is the benign or non-dangerous type, where no cholesteatoma is present. This type usually arises from a tubal or peritubal infection transmitted to the middle-ear cleft. The perforation of the drum-head is usually central or anterocentral, and the discharge, which may be continuous or intermittent is muco-purulent and odourless. Re-infection of the middle ear occurs either *viâ* the eustachian tube or *viâ* the external auditory meatus, largely by physical means and made possible by the presence of a physical defect in the tympanic membrane. Frequently this lesion is accompanied by an impairment of hearing, usually of the conductive type, which may become aggravated and progressively worse with each recurrent infection because of increased fibrosis or tympanosclerosis.

Secondly there is the dangerous type, cholesteatoma being present. The perforation is usually marginal involving Shrapnell's membrane or in the posterior segment where part of the bony rim has been destroyed.

* Owing to the indisposition of Dr. L. Verhoeven the lecture was delivered by J. D. K. Dawes.

The otorrhœa is offensively smelly, and is intermixed with epithelium and probably yellow in colour. Deafness may be mild or profound and usually of conductive type whereas in the destructive cases the deafness may be of perceptive or mixed type.

It is generally agreed that the term "cholesteatoma" is a misnomer for the suffix "-oma" suggests a neoplasm. However, the terms "cholesteatosis", "keratosis" or "epidermosis" are generally not accepted and for the purpose of this lecture the term "cholesteatoma" has been retained. Three major theories concerning the pathogenesis of cholesteatoma exist:

(1) The primitive cholesteatoma, which according to Virchow would be a congenital epithelial tumour arising from the ectoderm of the first branchial cleft. Any suppuration present would be of secondary origin. This theory has, for the present, been completely abandoned.

(2) The theory of epithelial metaplasia wherein Von Tröltsch, Politzer and Diamant maintain that the middle-ear mucosa undergoes a metaplastic change to become an epidermis-like layer under the influence of chronic pressure by pus. Lemâitre believes that the physio-pathological phenomena accompanying irritative or inflammatory conditions can change the tympanic mucosa into a stratified squamous epithelium. To-day this theory appears unlikely and is being abandoned by an increasing number of otologists.

(3) The theory of immigration wherein an extension of the stratified squamous epithelium from the external meatus occurs in an attempt to heal over the defects produced in the mucosa of the middle ear. Ruedi, Friedmann, Tumarkin, McGuckin and myself support this theory which was originally put forward by Bezold and Habermann.

A negative tympanic pressure produced by eustachian tube obstruction frequently occurs in children and this negative pressure may lead to a dry collapse of Shrapnell's membrane and œdema of the middle-ear mucosa. Secondary infection may arise in Prussak's space and so lead to the formation of an acute epitympanic polyp with an erosion of the outer attic wall. A careful examination of this early phase of cholesteatoma as seen most often in children has converted me to the immigration theory so well described by Alex Tumarkin in his article on "Attic Cholesteatosis" in August, 1958, in the *Journal of Laryngology*. This migration of cholesteatoma from the external meatus into the middle ear can be divided into 6 stages (Fig. 1).

1. (*a*) Dry collapse and perforation of Shrapnell's membrane.
2. (*b*) Secondary infection of Prussak's space with the formation

of an acute epitympanic polyp. At the same time an acute erosion of the adjacent outer attic wall may be noticed.

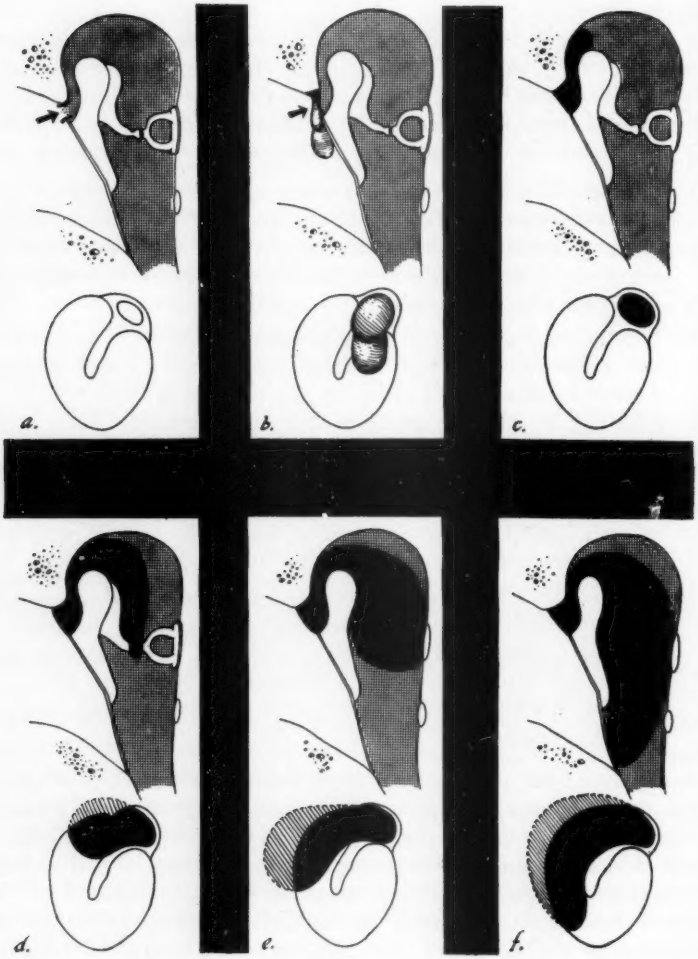

FIG. 1.
Diagram illustrating the stages of cholesteatomatous migration (see text).

3. (*c*) The early phase of cholesteatomatous migration through the attic. At this stage the ossicular chain functions normally and the pars tensa is intact.

4. (*d*) a deeper migration of cholesteatoma into the middle-ear cleft with necrosis of part of the ossicular chain. The pars tensa may remain intact or itself be perforated by the migrating cholesteatoma. The lenticular portion of the long process of the incus is usually the first bony part to necrose probably due to interference with its precarious blood supply by the cholesteatoma.
5. (*e*) An extensive migration of the cholesteatoma into the middle ear producing necrosis of the ossicular chain and perforations in both the flaccid and tense parts of the tympanic membrane but without intracranial complications. Generally the whole long process of the incus and the anterior and posterior crura of the stapes are eroded by disease.
6. (*f*) A similarly extensive invasion with intracranial complications.

This type of anatomical classification although useful is largely theoretical, for clinically there may be merging of the various stages, and intracranial complications may occur at any time.

The Natural Method of Repair of the Eardrum

Because of the large number of naturally healed perforations seen in otological practice my curiosity has been stimulated and the start, progress and end result of this spontaneous healing has been studied. It is well known that the ear-drum consists of an interstitial connective tissue layer covered by an epidermis outside and lined by middle-ear mucosa on the inside. Frequent careful microscopic examinations in the living have shown that the mucosal layer has the greatest blood supply and that it is this mucosal layer which plays the greatest part in spontaneous healing. Once the mucosal layer proliferates to close a gap the epidermis follows, whereas the interstitial layer never seems to regenerate. Thus the repaired part of the tympanic membrane consists of only mucosa and epidermis and consequently looks thinner and more transparent.

A year and a half ago, after a transmeatal suction clearance in a patient with a cholesteatoma occupying a posterior marginal perforation I was able to study the regeneration of the ear-drum. One week after this suction under 10× magnification I was able to see a little mucosal string stretching from the edge of the perforation to the hypotympanic space. With 16× magnification six mucosal strings could be seen and at 25× magnification 30 to 40 longitudinal and transverse strings were seen and the whole structure resembled a beautiful spider's web. Every week this regenerative process was carefully followed and after 6 weeks the perforation was healed by a two-layer drum which produced a spontaneous type III tympanoplasty and consequently a great improvement in the patient's hearing.

Following this, many cases of spontaneous regeneration of the ear-drum have been seen and studied and I have been surprised at the ease with which the tympanic membrane heals after a careful removal of disease when particular care has been taken not to disturb the local blood supply of the underlying mucosa.

[Seventeen coloured transparencies of ten ears were projected to illustrate tympanoplasties of various types produced by spontaneous healing. Two slides were also used to show that in spontaneous healing of a tympanic defect a mucosal web bridges the gap before the epithelium grows across.]

Having considered the natural history of chronic otitis in a simple manner we must proceed to the clinical study of the patient's problem. The first essential is the history of the disease as given by the patient. Let him tell you in his own way about the duration of the disease, the character of the discharge, whether it is continuous or intermittent, the degree and progress of his hearing loss, the discomfort or pain. At the same time assess the patient's intelligence and his reactions. A psychological leading question at the end of a complete examination should be "What is your main complaint and why have you come to see me now"?

A careful enquiry should be made into the patient's general health and in particular with regard to tuberculosis. I have found that in patients who have or have had active tuberculosis that there is a marked reduction in resistance to infection by chronic otitis and also a reduced efficiency in their powers of healing.

Head mirrors or Clar Lamps without the help of magnification are useless for a careful examination. To-day, an efficient otoscope with good focusing is the instrument of choice in the first instance. However, if there is any doubt concerning the integrity of the tympanic membrane, then the ear must be examined with a binocular microscope with the patient in a reclining position. A Zeiss otomicroscope should be always available in the consulting room. As a preliminary to careful examination under the microscope cleaning of the external meatus and middle ear by suction should be done. This may be very painful, and on the first occasion this examination is preceded by an injection of 0·5 to 0·8 ml. of 2 per cent. Xylocaine into the posterior meatal wall at the junction of the cartilaginous and bony portions. At this examination the following points should be observed.

(1) The size and location of any perforation.

(2) The condition of any drum remnants, the presence of adhesions, thickenings or calcium plaques.

(3) The character of the discharge.

(4) State of the mucosa, polyp formation or fibrosis, the presence of migrating squamous epithelium in the middle ear.

(5) If possible, the state and function of the oval and round windows, the ossicular chain, and the incudostapedial joint should be assessed, and the tympanic orifice of the eustachian tube inspected.

These findings should be described and a drawing made or better still a photograph taken.

Strange as it may seem, radiological studies of the temporal bone are of little help for their only value is to delineate the middle fossa dura or lateral sinus and to show the degree of pneumatization of the mastoid temporal bone. In general, it adds no more information than can be obtained by a careful examination of the ear.

Bacteriological examination, including sensitivity studies of the bacteria found in the aural discharge, appears to be of no value for the bacterial flora have been found to be inconsistent and frequently changing.

On the other hand, audiometric studies have frequently proved very useful. In those benign lesions with central or paracentral perforations, audiometry, carried out before and after a temporary closure of the perforation, has helped to determine the functional state of the ossicular chain. As early as 1841 James Yearsley was closing these central perforations with moist cotton wool to obtain improvements in hearing. Cotton wool moistened in paraffin, thin paper or polyethylene sheets have been used as a temporary measure for audiological studies. If the hearing improves after closure then we may conclude that the ossicular chain is working. However, if there is no improvement three possibilities must be considered; the ossicular chain is disrupted, or if intact it is fixed by tympanosclerosis, or the footplate of the stapes is fixed by otosclerosis.

On some occasions in the dangerous cholesteatomatous lesions it has been possible to assess the mobility of the footplate of the stapes by placing a temporary prosthesis over the gap and in contact with the head of the stapes.

Treatment using antiseptics or antibiotic powders or ear drops has been found to be valueless and indeed it is often contra-indicated for it inhibits spontaneous healing, or it produces an irritation of the middle-ear mucosa or external meatal skin and on occasion a crust formed of powder has shut in and obscured disease.

To-day more cases of mycotic infections of the external canal occur than ever before. Probably this is attributable to the abuse of local antibiotic ear drops.

Mr. J. H. Otty in his contribution to the discussion on the use and abuse of antibacterial drugs at the Section of Otology in April, 1960, made the following statement. "It is doubtful if antibacterial drugs or antibiotics have a place in the treatment of chronic otitis media. Antibiotics and antibacterial drugs should not be used by local application and I doubt the wisdom of giving small doses of sulphonamides or antibiotics over weeks, months or even years as a prophylactic since it may lead to organisms becoming resistant to these drugs."

Now, let us review the reasons that have led to the development of transmeatal suction clearance.

(1) There is a marked tendency to spontaneous healing in chronic otitis.

(2) The functional results of spontaneous healing are better than those following surgery.

(3) The use of antiseptic and antibiotic powders and drops and the syringing of the ear do not help to cure chronic otitis but rather disseminate infection and cholesteatoma throughout the temporal bone, breaking down the natural barriers of resistance and preventing regeneration of the drum and repair of the tympanum.

(4) Tympanoplastic surgery disturbs the blood supply of the ear and their late results are unsatisfactory. Many free postauricular skin grafts appear to desquamate and necrose through faulty circulation after six months or one to two years. A total skin graft of a mastoid cavity when laid on bare bone is not viable according to the plastic surgeons, and a "take" can only be expected over an area of 1 sq. cm. I do not think that a free skin graft normally exposed to the air can be expected to survive in a region of abnormal habitat such as the mastoid cavity where the temperature is higher.

(5) Since I believe that cholesteatoma is a migration from the external meatus, the disease in chronic otitis should be classed as accessible or inaccessible to an approach from the external auditory meatus. In many cases the cholesteatoma has been found to be not only accessible but also exciseable by a transmeatal approach with suction. In this minor procedure the external meatus and middle ear are cleaned with a straight suction cannula. Crusts and debris are detached using small serrated curettes and picked out with fine alligator forceps of Hartmann's type. Then the cholesteatoma together with its lining membrane are stripped from attic, aditus, and middle ear using very small excavators bent right or left, or are sucked out with varying sized sucker cannulae bent upwards. For cleaning the aditus, antrum and hypotympanum a suction cannula bent downwards is of great help. Small pieces of cholesteatoma, for example between the facial nerve and the stapes are carefully aspirated with fine needles of 0·6 to 1·2 mm. diameter. The degree of suction obtained being controlled by a valved thumbpiece attached to the needle.

On occasions, the cupped forceps of Zöllner are needed to remove granulation tissue, or fine scissors such as those of Bellucci, are used to divide fibrous bands and strands of tympanic membrane or to cut off the head of the malleus. These instruments are illustrated in Figs. 2-5.

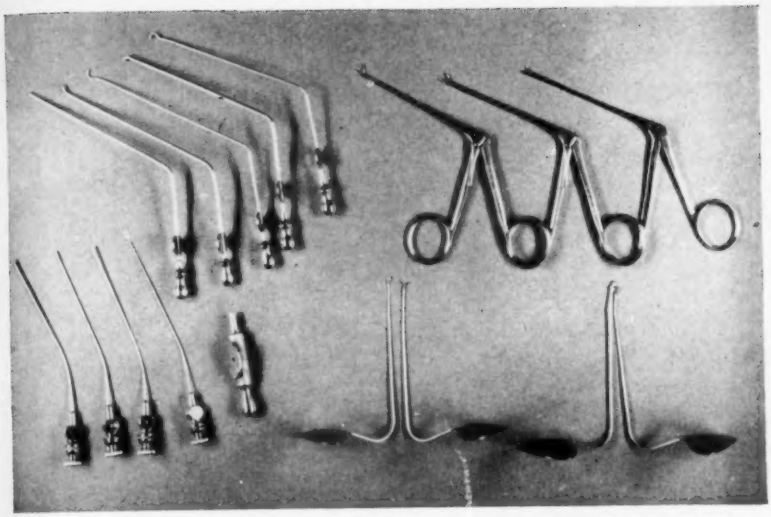

FIG. 2.
The instruments used in transmeatal suction clearance.

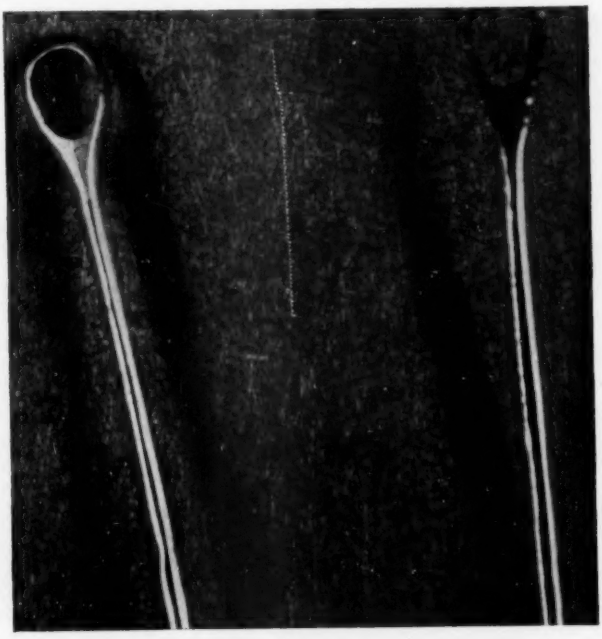

FIG. 3.
Enlarged views of fine serrated ring curettes.

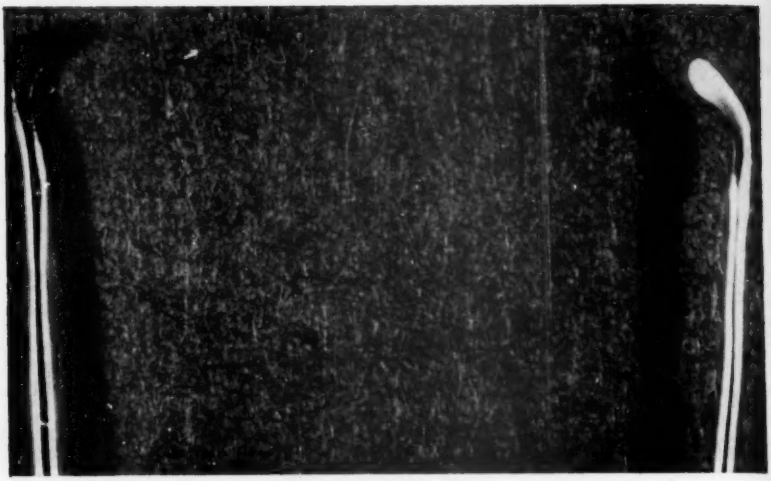

FIG. 4.
Enlarged view of dental excavators.

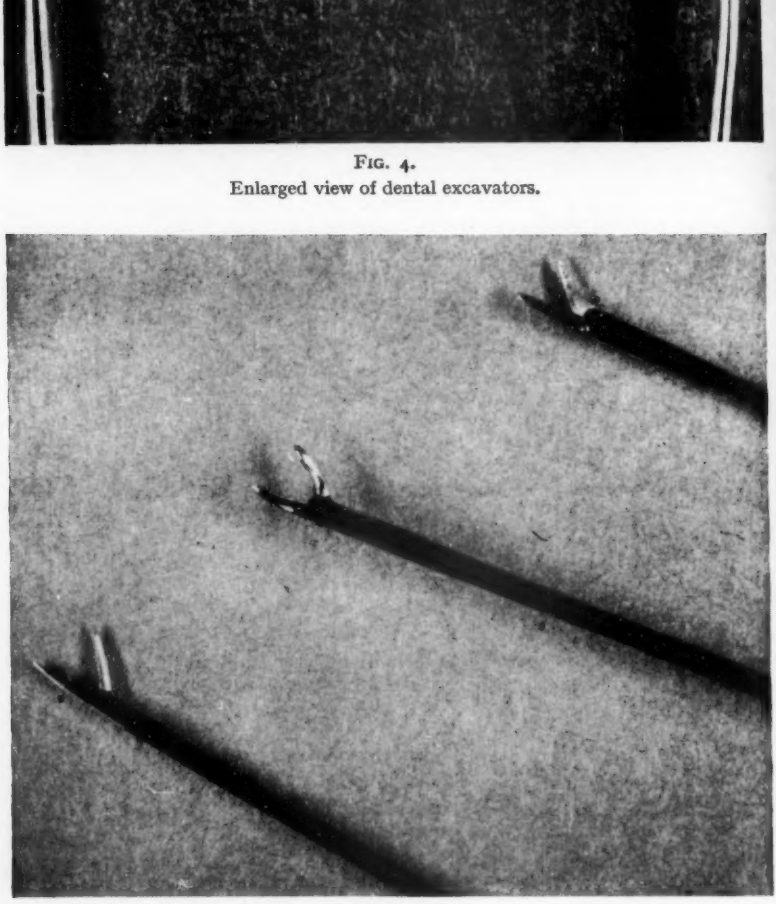

FIG. 5.
Enlarged view of Bellucci scissors, Zollner and Hartmann forceps.

A limited removal of outer attic and adital walls by curettes and drills can also be done if it is necessary to reach an extension. Large destructive lesions due to cholesteatoma with masses of granulation tissue cannot be cured by means of transmeatal suction for the infiltration is inaccessible. However, following the introduction of this method the number of cases submitted to tympanoplastic procedures has been reduced from 80 per cent. to 20 per cent. Nevertheless, some diseases still require a major surgical exploration.

A suction clearance is carried out at weekly intervals and after 2-6 suctions a cure is frequently obtained. The presence of small brown crusts suggests that recovery is well on the way.

[Thirteen coloured transparencies of six ears were used to illustrate the end result of transmeatal suction clearance.]

The prevention of cholesteatomatous destruction now seems possible. The recognition of the early state of migration by cholesteatoma under the operating microscope is possible and at this stage we should be able to cure the lesion before destruction of the middle-ear structures has occurred. Close co-operation with pædiatricians in the management of chronic otitis of more than 4 weeks' duration is necessary and each of these cases should be submitted to a careful microscopic examination under a general anæsthetic.

Acknowledgements

I would like to thank Mr. D. H. Hammersley of the Department of Surgery, University of Durham for drawing the diagram and also Miss D. Forster for typing several scripts for Mr. J. D. K. Dawes.

4 Nervierstraat,
Antwerp,
Belgium

AN EXPERIMENTAL STUDY OF THE INFLUENCE OF THE ENDOCRINE SYSTEM ON THE NASAL RESPIRATORY MUCOSA

By MILES TAYLOR (Sheffield)

THERE is considerable clinical and experimental evidence to indicate that the endocrine system influences the nasal respiratory mucosa (Taylor, 1959). The manner in which it does so is obscure. In other tissues it is known that some biochemical processes are under hormonal control (Krebs, 1956), and in view of the clinical and experimental evidence it would be reasonable to suggest that a similar hormonal-control mechanism exists for the nasal mucosa. The present work is an attempt to investigate this relationship by experimental means. Histochemical methods have been used to indicate some of the chemical substances that occur within the cells of the nasal mucosa. The effect of the endocrine system on the distribution of these substances has been studied by performing these histochemical tests in the rat after the removal of endocrine glands, and after the injection of hormones; and in the rabbit during pregnancy and after the implantation of hormones.

Materials and Methods

A total number of 220 animals was used, and the following table indicates the type of operation and the number of animals used in each experimental procedure.

	Rat	*Control*
Oöphorectomy	10	5
Orchidectomy	10	5
Thyroidectomy	10 (5 male, 5 female)	5 (2 male, 3 female)
Hypophysectomy	10 (5 male, 5 female)	5 (3 male, 2 female)
Adrenalectomy	20 (10 male, 10 female)	5 (2 male, 3 female)
Injection of Oestradiol ..	20 (10 male, 10 female)	5 (3 male, 2 female)
Injection of Progesterone ..	20 (10 male, 10 female)	5 (2 male, 3 female)
Injection of Testosterone ..	20 (10 male, 10 female)	5 (3 male, 2 female)
	Rabbit	
Pregnancy	25	5
Implantation of Oestradiol ..	10 (5 male, 5 female)	5 (2 male, 3 female)
Implantation of Progesterone	10 (5 male, 5 female)	5 (3 male, 2 female)

The operative procedures and post-operative care were carried out according to the methods recommended by Griffith and Farris (1942) and

the animals sacrificed after six weeks, with the exception of the rabbits in the pregnancy experiments. As the period of gestation in the rabbit is 30 days, groups of 5 animals were killed at 5, 10, 15, 20 and 30 days respectively.

The implants of œstradiol and progesterone, in the form of cylinders 5 mm. long and 2 mm. in diameter, and composed of nine parts of cholesterol and one part of hormone, were implanted subcutaneously in the anterior abdominal wall. Each implant weighed 25 mg. and thus contained 2·5 mg. of hormone. In each rabbit the bulk of the implant was recovered when the animal was killed after 6 weeks and the average rate of absorption was found to be of the order 2-4 μg/day. This corresponded closely with the information supplied by the manufacturers, who stated that the daily absorption of œstrogen and progesterone in the form supplied was about 3 μg/day.

Reynolds and Foster (1940) determined by a physiological method the acetylcholine-equivalent content of nasal mucosa before and after the administration of œstrogen. They injected 50-2,000 μg of œstradiol intramuscularly in a series of animals and found that a maximum response occurred after 1 hour. This procedure was adopted in the present study using progesterone and testosterone as well as œstradiol.

The part of the nasal septum bearing respiratory mucosa on each side was removed from the animal immediately after death. Each specimen was divided into two, one part being fixed in ice-cold acetone and the other in ice-cold 80 per cent. alcohol. After fixation the mucosa was stripped from the septa and embedded in paraffin wax. As the mucosa tended to fragment rather easily during sectioning a number of specimens were fixed by the method of freeze substitution. Although it gave a better histological picture with more accurate localization of intracellular substances, it was not used routinely as it had the disadvantage that the mucosa became difficult to strip from the septa.

Apart from routine staining with hæmatoxylin and eosin, the following stains were used.

(1) Iron hæmatoxylin and picrofuchsin (van Gieson) and Masson's trichrome for the determination of collagen.

(2) Gordon and Sweet's stain for reticulin.

(3) Southgate mucicarmine for mucins.

(4) Alcian blue for acid mucopolysaccharide.

(5) The periodic acid-Schiff (P.A.S.) method (Hotchkiss, 1948) for the demonstration of polysaccharides. In some instances the acid reducing rinse was not employed; this enhances the demonstration of collagenous tissue, as suggested by Lillie (1953).

(6) Toluidine blue (0·5 per cent. for 30 minutes) and azure A (0·01

per cent. for 5 minutes or 0·1 per cent. for 10 minutes) to demonstrate metachromasia.

(7) Toluidine blue (0·5 per cent. for 30 minutes) and the methyl green pyronin method for ribonucleic acid (R.N.A.). In the latter technique, pyronin Y was used as advocated by Kurnick (1955). Control sections were stained after treatment overnight with 10 per cent. perchloric acid at 4° C. (Seigel and Worley, 1951).

(8) The Feulgen reaction to demonstrate deoxyribonucleic acid (D.N.A.) (Pearse, 1960).

(9) The α-napthol acetate method (Pearse, 1960) for non-specific esterase.

(10) The myristoyl choline method (Pearse, 1960) for cholinesterase.

(11) The calcium-cobalt method of Gomori (1946) for alkaline phosphatase with the addition of magnesium ions to activate the enzyme as suggested by Kabat and Furth (1941). The substrate used was sodium β-glycerophosphate and the incubation period 1 hour.

Results

There were no morphological or histochemical changes in the nasal mucosa of the rat following orchidectomy; oöphorectomy; thyroidectomy; hypophysectomy; the injection of progesterone; or the injection of testosterone: nor in the rabbit after the implantation of progesterone.

The normal appearance in these animals has already been described by Taylor (1958).

There were no naked eye changes in the nasal mucosa following bilateral adrenalectomy in the rat, but histochemical examination showed that there was an increase in the amount of alkaline phosphatase. It lay mainly in the superficial parts of the cytoplasm of the ciliated epithelial cells and was evenly distributed. It was not a constant finding, occurring in seven out of ten male, and five out of ten female rats. There were no other changes in the histochemical characteristics of the mucosa.

To the naked eye there appeared to be an increase in the thickness and vascularity of the nasal respiratory mucosa of the rabbit during pregnancy. It was more marked in the second half of pregnancy. This impression was confirmed on histological examination of the mucosa. Numerous dilated blood containing spaces were seen deep to the basal cubical layer of cells. There were no other histological or histochemical changes in the nasal respiratory mucosa.

After the implantation and injection of œstradiol the nasal respiratory mucosa was thicker and more vascular than in the normal rat or rabbit. Histological examination showed that in both series there were numerous dilated blood containing spaces below the basal cubical layer of cells.

No other histological or histochemical changes were seen in the nasal respiratory mucosa.

Discussion

The vasodilator effect of œstrogens on the nasal mucosa of the rat has been described previously by Zanzucchi (1937) and Rudolfo-Masera (1940), and the present work confirms their findings. Similar effects on the nasal mucosa of man and monkey were described by Conrad (1936), Mortimer, Wright, Bachman and Collip (1936), Mortimer, Wright and Collip (1936) and Mennega and Tausk (1941). Reynolds and Foster (1940) believed that this was due to a decrease in acetylcholinesterase activity, but it has not proved possible to verify this in the present work, since no acetylcholinesterase was discovered in the nasal mucosa of the rabbit or the rat, before or after the administration of œstradiol, or during pregnancy in the rabbit.

In the present study, no changes in the nasal mucosa have followed the removal of the ovaries or testes of adult rats. This does not agree with the work of Champny and Kritch (1929), Borghesan (1931), or Pighini and Porta (1934) who found that degenerative changes occurred in the nasal mucosa following orchidectomy. It has been shown that degenerative changes occur quite commonly in the nasal mucosa of intact "stock" rats (Kelemen and Sargent, 1946), guinea-pigs (Kelemen, 1950) and rabbits (Kelemen, 1955). In the present work two series of experiments were invalidated because the nasal mucosa of the control animals showed degenerative changes. Adequate controls are therefore essential, as without them reports of changes in the nasal mucosa must be accepted with caution. Borghesan (1931) also reported a case of degeneration of the nasal mucosa following oöphorectomy in a young woman. This supports the work of Wustrow (1951) who showed that atrophic changes occurred in the cavernous tissue spaces with the onset of sterility in man.

The species differences may then be due to anatomical factors, as there is no cavernous tissue in the nasal mucosa of the rat, and this is probably sufficient to account for the differences between the nasal mucosa of the rat and that of man and monkey following the injection of testosterone propionate. In the former no changes were seen, while gross congestion and swellings of the nasal mucosa with perivascular œdema was a marked feature in man and monkey (Hamilton, 1937; Tebbutt, 1938).

In the present study no macroscopic or histological changes were observed in the rat following thyroidectomy. Although the findings of Pearlman, Kahn and Kunde (1929) and Chavanne (1936) were inconclusive, Proud and Lange (1957) described swelling and pallor of the nasal mucosa with loss of cilia as a constant finding in the dog. This agrees with the clinical observations of Proetz (1950), but many of his patients had a red, dry and "chapped" nasal mucosa and not a pale "soggy" one.

There appears to be an increase in the amount of alkaline phosphatase

in the cytoplasm of the ciliated epithelial cells of the nasal mucosa of the rat following adrenalectomy. As it was not a constant finding it may be of no significance, but its presence here is unexpected. In epithelia such as those in the intestine and kidney, the presence of phosphatase is usually associated with absorption, and although it occurs in other situations, such as ossification centres, no reason can be suggested for its existence in the site demonstrated here.

The results that have been obtained are mainly negative. This may be due to anatomical differences between the nasal mucosa of man and the rabbit and rat, or to a lack of sensitivity of the histochemical tests used. Thus biochemical assays or more refined histochemical techniques may be of value in elucidating some of the problems raised in this work. For example: the mode of action of œstrogens on the nasal mucosa appears to be relevant to an understanding of nasal physiology; and in particular, why œstrogens are concentrated in the nasal mucosa, and how they reduce the level of functioning acetylcholinesterase. The role the pituitary gland plays in this mechanism is also obscure and merits further study. Thus although the hormonal control of biochemical processes is now well established, the available evidence seems to indicate that the endocrine system influences the nasal respiratory mucosa mainly through its effect in determining the level of functioning acetylcholine.

Summary

The histological and histochemical features of the nasal respiratory mucosa of the rabbit and rat have been examined after the removal of endocrine glands, and after the administration of hormones and during pregnancy. There appears to be an increase in the size of vascular spaces in the rabbit and rat after the administration of œstrogens, and in the rabbit during pregnancy, otherwise no histological or histochemical changes were noted, with the exception of an increase in alkaline phosphatase in the epithelia of the mucosa of some of the rats after adrenalectomy.

No evidence has been produced to show that the histochemical characteristics of the nasal mucosa in the rabbit and rat are affected by hormonal control although it must be borne in mind that histochemical tests give a limited indication of the metabolic processes taking place in this tissue.

Acknowledgements

I wish to express my thanks to Professor F. Davies for his advice and encouragement during the carrying out of the research reported here, and for the facilities he has made available in his Department in the University of Sheffield. I am indebted to Mr. Austen Young for his encouragement and interest, to Dr. R. M. H. McMinn for his advice and criticism, and to Mr. J. H. Kugler and Mr. J. H. Morrill for their technical advice and assistance.

REFERENCES

BORGHESAN, E. (1931) *Valsalva*, **7**, 625, 646.
CHAMPNY, C., and KRITCH, N. (1929) *C.R. Soc. Biol.* (*Paris*), **100**, 413.
CHAVANNE, L. (1936) *Oto-rhino-laryng. int.*, **20**, 653.
GOMORI, G. (1946) *Amer. J. clin. Path.*, **16**, *Tech. Sect.*, **6**, 177.
GRIFFITH, J. Q., and FARRIS, J. F. (1942) "The Rat in Laboratory Investigation". Philadelphia.
HAMILTON, J. B. (1937) *Proc. Soc. exp. Biol.* (*N.Y.*), **37**, 366.
HOTCHKISS, R. D. (1948) *Arch. Biochem.*, **16**, 131.
KABAT, E. A., and FURTH, J. (1941) *Amer. J. Path.*, **17**, 303.
KELEMEN, G., and SARGENT, F. (1946) *Arch. Otolaryng.* (*Chicago*), **44**, 24.
—— (1950) *Arch. Otolaryng.* (*Chicago*), **52**, 579.
—— (1955) *A.M.A. Arch. Otolaryng.*, **61**, 497.
KREBS, H. A. (1957) *Brit. postgrad. med. Fed.: Lectures on the Scientific Basis of Medicine* (*1955-56*), **5**, 126.
KURNICK, N. B. (1955) *Stain. Technol.*, **30**, 213.
LILLIE, R. D. (1953) *J. Histochem. Cytochem.*, **1**, 353.
MENNEGA, A.M.W., and TAUSK, M. (1941) *Arch. int. Pharmacodyn.*, **66**, 26.
MORTIMER, H., WRIGHT, R. P., BACHMAN, C., and COLLIP, J. B. (1936) *Proc. Soc. exp. Biol.* (*N.Y.*), **34**, 535.
MORTIMER, H., WRIGHT R. P., and COLLIP, J. B. (1936) *Canad. med. Ass. J.*, **35**, 503.
PEARLMAN, S. J., KAHN, N. E., and KUNDE, M. M. (1929) *Trans. Amer. laryng. rhin. otol. Soc.*, **35**, 337.
PEARSE, A. G. E. (1960) "Histochemistry" 2nd edition. London.
PIGHINI, G., and PORTA, C. F. (1934) *Valsalva*, **10**, 140.
PROETZ, A. W. (1950) *Laryngoscope* (*St. Louis*), **60**, 627.
PROUD, G. O., and LANGE, R. D. (1957) *Laryngoscope* (*St. Louis*), **67**, 201.
REYNOLDS, S. R. M., and FOSTER, F. I. (1940) *Amer. J. Physiol.*, **131**, 422.
RUDOLPHO-MASERA, T. (1940) *Arch. ital. Otol.*, **52**, 525.
SEIGEL, G. B., and WORLEY, L. G. (1951) *Anat. Rec.*, **111**, 597.
TAYLOR, M. (1959) M.D. Thesis. No. 1066. University of Sheffield.
TEBBUTT, H. K. (1938) *N.Y. St. J. Med.*, **38**, 332.
WUSTROW, F. (1951) *Z. Anat. Entwickl. Gesch.*, **116**, 139.
ZANZUCCHI, G. (1937) *Arch. ital. Otol.*, **49**, 255.

Department of Anatomy,
The University,
Sheffield 10

DEAFNESS AND THE TREACHER COLLINS SYNDROME

By C. A. HOLBOROW (London)

Two cases of congenital deafness due to absence of the incus have been seen recently in related patients showing to a minor degree the abnormalities of the Treacher Collins syndrome. In each case a satisfactory gain in hearing followed tympanotomy and insertion of a polythene prosthesis.

Case Histories

Mr. L.P. (Figs.1(*a*) and 1(*b*)), a builder's labourer aged 36, attended Guy's Hospital on August 31st, 1960, with a history of very long-standing deafness in both ears. There was no history of other aural trouble apart from some discharge from his right ear during the course of "electrical treatment" for his deafness. There was no paracusis or tinnitus and, at first, he denied any deafness in other members of his family. A hearing aid had been provided at another hospital but he made no use of it.

Examination showed no abnormality of his nose or throat. Both externa auditory meatuses were very sloping and there was a healed perforation of the right tympanic membrane. The left tympanic membrane was normal. An audiogram (Fig. 3) was taken and showed a severe bilateral conductive deafness with good bone conduction. Though not a typical case, the patient was thought to suffer from otosclerosis and he was placed on the waiting list for a left stapes mobilization procedure.

At operation on November 16, 1960, under local anæsthesia, a left tympanotomy showed that the incus was completely absent (Fig. 7: L.P. 1). The stapes was flattened and deformed but it was mobile and a round window reflex was easily obtained. No stapedius tendon was present but there was a thin strand of fibrous tissue, possibly representing the incus, running from the neck of the stapes to the malleus. A polythene prosthesis was fashioned of such a length that when it was fixed over the head of the stapes the tympanic membrane would be in contact with the other end (Fig. 7: L.P. 2). The strand of fibrous tissue was severed and the tympanic membrane replaced. There was an immediate gain in hearing.

Convalescence was uneventful and a post-operative audiogram (Fig. 4) showed a most satisfactory gain in hearing which has been maintained. Before he left hospital the patient now mentioned that he had a sister who was also deaf and who wished to attend for possible surgery. In addition, he asked that his right ear should undergo a similar operation at a later date.

The sister of this patient attended Guy's Hospital on December 20th, 1960. She was Mrs. E.R. (Figs. 2(*a*) and 2(*b*)), a housewife aged 31, married with one

FIG. 1(*a*). FIG. 1(*b*).

L.P., aged 36 years. Depressed malar below and lateral to external canthus. More marked on the right side.

FIG. 2(*a*). FIG. 2(*b*).

E.R., aged 31 years. Sloping palpebral fissures. Ptosis auriculae. Depression of malar bones especially on the left side.

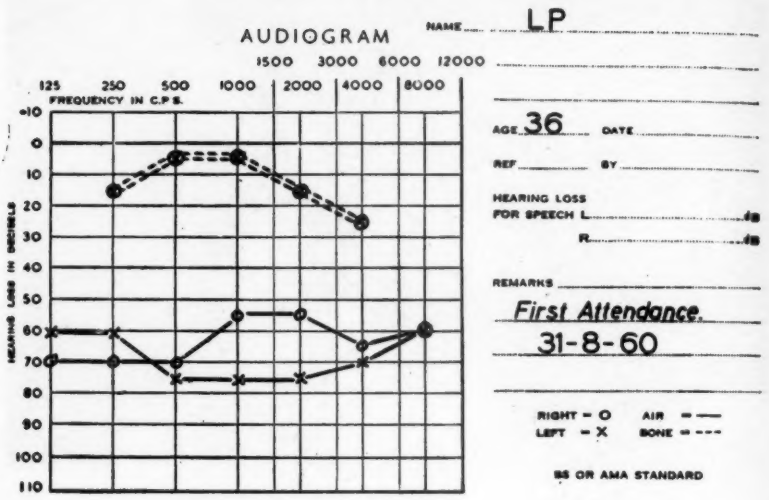

FIG. 3.
Audiogram. L.P. First attendance. August 31st 1960.

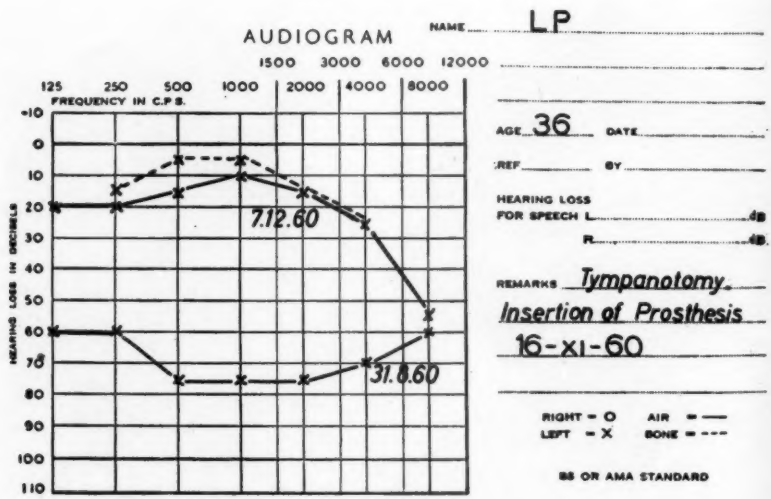

FIG. 4.
Audiogram. L.P. Left ear. Before and after surgery.

daughter. She gave a history of deafness in both ears for as long as she could remember. The degree of deafness had never altered and there had been no change after the birth of her daughter, now aged 8. There had been no aural discharge, tinnitus or vertigo but she had noticed marked paracusis. She made good use of a hearing aid which had been supplied by another hospital where she was under treatment for ulcerative colitis.

Examination showed a pleasant woman with somewhat unusual features. The palpebral fissures sloped slightly downwards and outwards and the lower lids were devoid of lashes over their medial two-thirds. The malar bones were "dented" below and lateral to the external canthi. There was some asymmetry of her chin. Her nose and pharynx were normal. Her ears were small and were placed lower than normal. The external auditory meatuses were narrow especially on the right side, and sloped markedly. The tympanic membranes were normal in appearance, though the right drum could not be seen completely. Voice and tuning fork tests showed that she had a severe bilateral conductive deafness and she was placed on the waiting list for tympanotomy.

At this point it was observed that she had some of the characteristics of the Treacher Collins syndrome. It was then, as always, easy to be wise after the event and to remember the slightly flattened malar bones and sloping meatuses of her brother. A careful family history, however, provides no other case of deafness; these two patients were the only deaf members of a family of four brothers and two sisters. The father and mother were normal as was the 8-year-old daughter of Mrs. E. R.

She was admitted on January 4th, 1961. An audiogram taken at that time (Fig. 5) showed a conductive deafness similar to that of her brother. In view of the difficulty of access to the right middle ear, a left tympanotomy was decided upon despite the better hearing over the speech range on that side.

At operation on January 5th under local anæsthesia, it was little surprise to find the incus absent (Fig. 7: E.R. 1). The stapes was represented by a small deformed bone without separate crura. It was mobile and a round window reflex could be obtained. In this case the tendon of stapedius was present and appeared to be normal. A polythene prosthesis was fashioned to connect the tympanic membrane to the stapes and, again, there was an immediate improvement in hearing (Fig. 6).

Soon after returning home she had a cold and found it necessary to use her aid for a few days. When this cleared up she discarded her aid completely. She now thinks she hears well enough and does not wish to undergo any further operation for the time being.

Comment

The Treacher Collins syndrome (mandibulo-facial dysostosis) was described in 1900. Since that time many authors have reported cases and the associated deformities comprising the syndrome have multiplied. These vary considerably and may occur, on one side or both, in different combinations. A comprehensive list has been given in this Journal (Spencer Harrison, 1957). The most common affect the ears, eyes, malar bones and mandibles and are illustrated, to a minor degree, by these two patients.

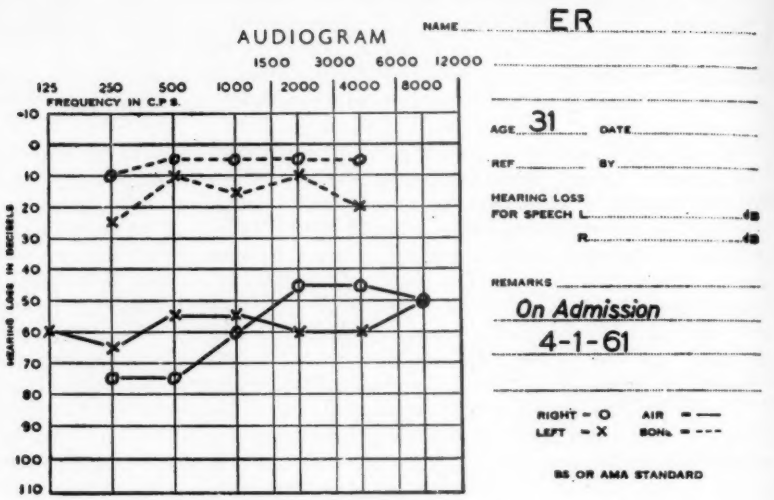
AUDIOGRAM
NAME ER
1500 3000 6000 12000
125 250 500 1000 2000 4000 8000
FREQUENCY IN C.P.S.
HEARING LOSS IN DECIBELS
-10 0 10 20 30 40 50 60 70 80 90 100 110
AGE 31 DATE
REF BY
HEARING LOSS FOR SPEECH L dB
R dB
REMARKS
On Admission
4-1-61
RIGHT = O AIR = ——
LEFT = X BONE = - - -
BS OR AMA STANDARD

FIG. 5.
Audiogram. E.R. On Admission. January 4th, 1961.

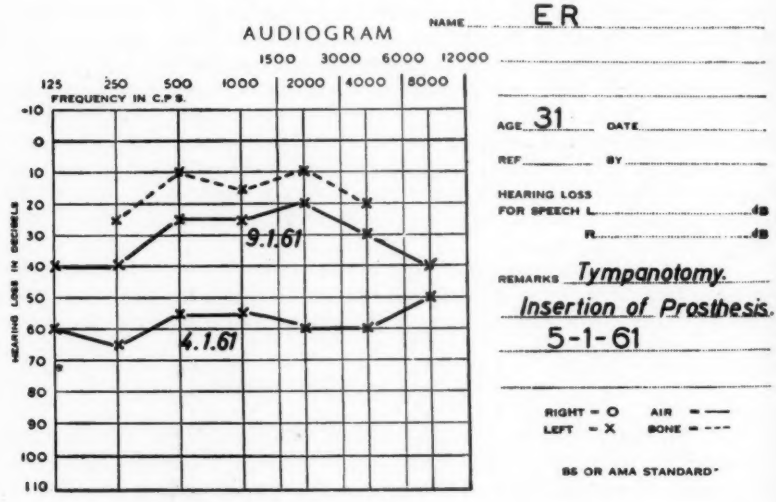

FIG. 6.
Audiogram. E.R. Left ear. Before and after surgery.

Cases tend to be found among the lower social and economic groups. The syndrome is congenital and its familial nature is illustrated though only two out of six siblings were affected.

Ears: Mrs. E. R. shows ptosis auriculae and both she and her brother have deformed external auditory meatuses which are narrowed and

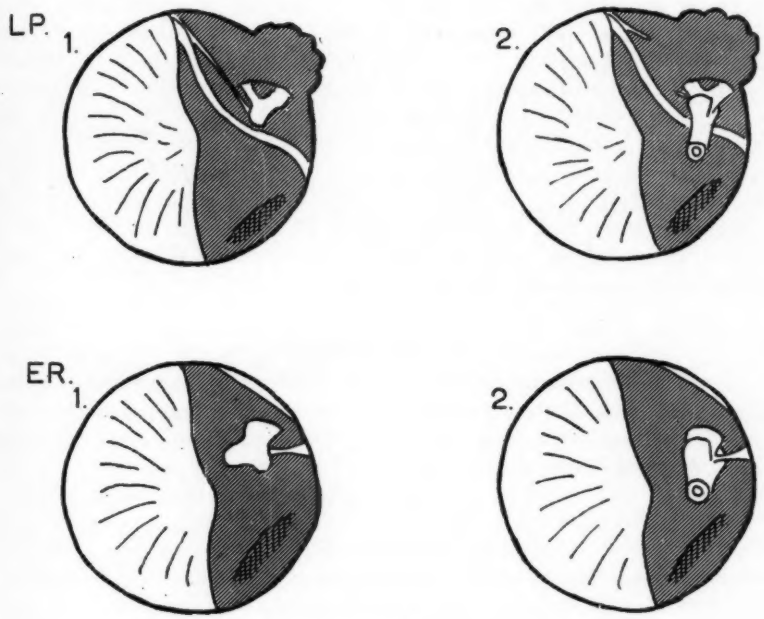

FIG. 7.
Diagrams showing findings at operation.

sloping. More severe cases may show microtia and meatal atresia. Malformations of the middle ear are common and it is probably more usual to find absence of middle-ear cleft or gross malformations of the ossicles with fusion of the malleus and incus (Livingstone, 1959). Deformities of the inner ear occur less frequently. I can find no other record of simple absence of the incus in the presence of an apparently normal malleus and tympanic membrane.

Eyes: the characteristic facial appearance of individuals with this syndrome is chiefly due to the formation of the palpebral fissures. These slope downwards and outwards and there is notching of the lower lid. Mrs. E.R. shows a suggestion of this notching together with another feature; absence of eyelashes over the medial portion of the lower eyelid.

Malar bones: flattening of the malar bones is clearly seen in both these patients.

Mandibles: micrognathia is common but is not seen here. There is, however, a suggestion of hypoplasia of the left mandible of Mrs. E.R.

These patients are of normal intelligence. Children showing the strange facial appearance of this syndrome are, however, often thought to be mentally defective and this mistaken impression is strengthened when they are deaf as well. Treatment of this deafness is, therefore, of great importance and as it is often of a conductive type it may respond to surgery. In cases with meatal atresia and gross abnormalities of the middle ear extensive operations have been described (Livingstone, 1959). It was fortunate that in these two patients a simple procedure resulted in such satisfactory hearing gain. One disadvantage of the procedure described would seem to be the possibility that the polythene tube might ulcerate through the tympanic membrane against which it rests. In the event of this accident further surgery could be undertaken as there has been minimal interference with the middle ear.

Acknowledgements

I wish to express my gratitude to Mr. Philip Reading, Surgeon to the Ear and Throat Department, Guy's Hospital, for permission to treat and record these cases. I am indebted to the Photographic Department at Guy's Hospital for the photographs.

REFERENCES

Collins, E. Treacher (1900) *Trans. ophthal. Soc. U.K.*, **20**, 90.
Harrison, M. Spencer (1957) *J. Laryng.*, **71**, 597.
Livingstone, G. (1959) *J. Laryng.*, **73**, 231.

Guy's Hospital,
London, S.E.1

THE USE OF LYOPHILIZED PLASMA SUBSTANCE IN TYMPANOPLASTY

By G. RÉVÉSZ (Budapest)*

ABSORBABLE foreign substances are often used with tympanoplasties, in order to support for a time the implanted free skin graft, to facilitate the creation of an air-holding cavity and aerization of the tympanic cavity from the tuba. According to data in the literature some authors tried to keep the skin graft in place by creating a direct contact between the flap thicker in its central part and the previously scarified promontorial mucosa. However, the functional results of such procedures were poor because of the extensive scar formation and adhesions. Thus, according to the view put forward by most workers and shared by us, too, no effort should be spared to keep the graft away from the mucosa of the medial wall of the tympanum and to prevent the development of scars.

One feels rather reluctant to claim that the various fibrin or gelatine preparations (Fibrinfoam, Fibrospum, Fibrostan, Spongostan, Oxycel, Sorbocel, Spongel, Calgitex, etc.) have fully fulfilled the hope attached to them. The problems raised by these preparations are usually twofold: the cellular reaction elicited by them and how they are absorbed.

Of course, the cellular tissue-reaction depends on the kind of the implant, it is usually smaller with the gelatine preparations. Considering that with every sort of operation involving transplantation of tissues and especially with tympanoplasties, it is essential that the cellular reaction should be the smallest possible, we strive to create the most favourable conditions for the healing of the flap. In what measure the accumulation of granulocytes, fibroblasts and plasma cells soon following the implantation of fibrin or gelatine, then the appearance at a later stage of lymphocytes, giant cells and eosinophils, and an eventual formation of excess discharge may be considered physiological or pathological, it is not primarily and not exclusively a theoretical question. The formation of a larger amount of fluid under the graft may interfere with the "take" of the flap and may endanger its viability.

This problem does not seem to have been solved by the addition of pantothenic acid (Bepathen) to increase tissue resistance, cortisone or hydrocortisone and antibiotics to ameliorate or prevent inflammation to the grafts.

* From the Health Service of the Hungarian People's Army.

Although some authors mention that gelatine preparations, in the first place, seem to be more rapidly absorbed, it should be borne in mind that for example Frenckner has demonstrated the presence of Oxycel residues as late as 4 to 5 months after operation. Richtnèr is quite firm in his opinion that whenever it is possible foreign substance implants should not be used in the middle ear, because they may not become absorbed and thus may impair hearing.

In addition to the technical, mechanical viewpoints mentioned, the substance introduced into the tympanum has a rôle to play in the nutrition of the flap as well. I intentionally do not wish to deal here with the question of blood supply from the recipient area, wishing to focus attention exclusively on the free-lying part of the flap, the one which is most critical from the point of view of nutrition. Unlike the fibrin preparations, gelatine, an incomplete protein product of partial collagen hydrolysis, is caused to dissolve by enzyme and phagocytic action and with its amino-acids takes part in the nutrition of the flap.

According to Conway the survival of the flap depends on the extracellular nutrition through imbibition. To be more precise: Paget and Conway have shown that during the first days plasma is flowing out from the blood vessels of the recipient area and enters the flap by diffusion.

To solve the above outlined problems of tympanoplastic operations, I have used a material that lends suitable support to the flap and keeps it elevated until fixation or tamponade is applied, that is absorbed without residue from the tympanum and that also takes part in the nutrition of the graft. I thought to have found this material in the lyophilized plasma substance. As it is known, lyoplasma is dissolved completely within one or two minutes in blood, saline solution, tissue fluids, water, etc. Being rich in proteins, carbohydrates and lipids, it is a direct source of nutritive compounds for the graft.

Since the dry lyoplasma substance is dissolved extremely rapidly, in this form it is not suitable for use as a support to the graft. It may become adequately firm, however, if treated by suitable chemical or physical processes.

Formaldehyde is a chemical known to possess a solidifying action and this degree of formolization is proportionate to the duration of pre-treatment. In spite of the favourable, desantigenization-inducing action of the formaldehydecondensation of proteins, this method had to be rejected. Formaldehyde may combine with protein or with the free amino-acid groups as, (1) free, (2) adsorbed, and (3) bound formaldehyde. Although the amount of free formaldehyde resulting from formaldehyde vapour pre-treatment may be minute, it still rules out this method as one of choice.

As it has been proved by several statistics, tympanoplasties may, in a certain proportion of the cases be followed by hearing loss of the perceptive

type, explicable in part by mechanical factors, such as manipulations in the area of the fenestrae, toxins and chemical agents (e.g. streptomycin). It is therefore possible that formaldehyde may enter the internal ear (even if in microgram amounts only) through the fenestrae, causing destruction therein. We have therefore not even attempted to employ this method.

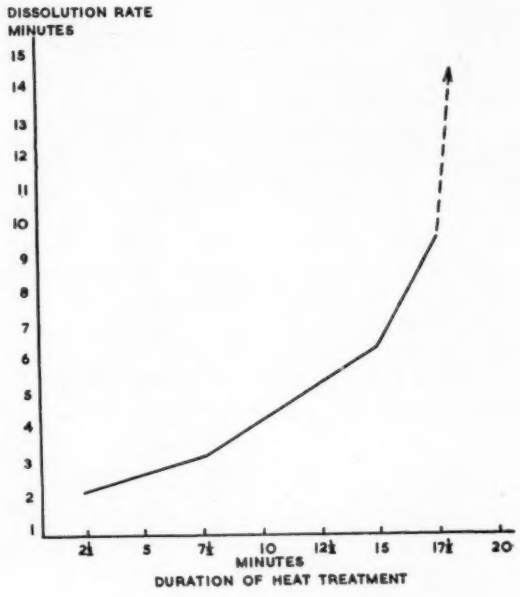

FIG. 1.

The physical method by which the lyoplasma substance may be hardened is heat treatment. The material, cut up into cubicles of 1 cubic cm. in size each, was exposed to dry heat of 120° C. for various lengths of time. The cubicles thus treated were then tested for solubility in 2 ml. volumes of 37° C. distilled water, physiological saline solution and serum. As shown by Fig. 1, solubility rapidly deteriorates with the duration of heat treatment; after 19 minutes of exposure the substance turns brown and is no longer soluble in the above-mentioned solvents.

For our purposes, heat treatment for 15 to 17½ minutes, proved to be the best, after which dissolution began in about 10 minutes and was complete in about 20 to 25 minutes. This gives ample time for the skilled operator to place the graft or to employ tamponade.

As it has been mentioned, the lyoplasma substance we used turns a

deepening yellow during heat treatment, turning suddenly brown at 19 minutes, obviously due to the so-called Maillard reaction, which is understood to mean the formation of so-called "brown substances" through a combination of carbohydrate components with amino-acids, peptides and the amino components of proteins. It is plausible that this is what happens, because the lyoplasma is prepared from blood stabilized with the ACD stabilizer, that contains as much as 3 g. per cent. of dextrose. The labile aldosylamines formed in the course of the Maillard reaction are rapidly converted to insoluble, brown compounds. This explains the solubility curve obtained after heat treatment.

We have been using the lyoplasma substance for years, with very good results. A pre-condition to its practical use is that the tympanum should be possibly dry when we place the lyoplasma substance. A small amount of moisture does not interfere with the results but makes it difficult to place the substance, by means of a dry spoon or pincets. The skin flap of suitable size is submerged 2 or 3 minutes in plasma solution; after it is placed a solution of thrombin is dripped on its margins and thus the flap is "glued" to the recipient area. The flap becomes fixed in place in 17 seconds and thus it will not become dislodged.

Finally, I should like to express my thanks to the National Blood Donor Service and to Dr. M. Gerendás, for the donation of the experimental material and for valuable advice, respectively.

Summary

To support the flap graft in tympanoplasty, a lyoplasma substance is used. It is more perfectly absorbed than are the materials used previously and until it becomes absorbed it serves as a source of nutritive materials for the unvascularized grafts.

REFERENCES

ALBRITTON, E. C. (1952) "Standard Values in Blood". Philadelphia.
BEALES, P. H. (1957) *J. Laryng.*, **71**, 162.
BORIANI, V.A., and OTTAVIANI, A. (1958) *Minerva otorinolaring.*, **9**, 337.
FASOLD, H., and TURBA, T. (1959) "Dtsch. Ges. Ernährung E. V. Symposion". München.
FRENCKNER, P. (1957) *Acta oto-laryng.* (*Stockh.*), **48**, 277.
GUILFORD, F. R., and WRIGHT, W. K. (1958) *Laryngoscope* (*St. Louis*), **68**, 825.
GUILFORD, F. R., WRIGHT, W. K., and DRAPER, W. L. (1959), *A.M.A. Arch. Otolaryng.*, **69**, 70.
HEILMEYER, L. (1960) "Lehrbuch der speziellen pathologischen Physiologie". Stuttgart.
HEYNS, K., and PAULSEN, H. (1959) "Dtsch. Ges. Ernährung E. V. Symposion". München.
HOUSE, L. R. (1958) *Laryngoscope* (*St. Louis*), **68**, 1481.

KLEY, W. (1954) *Z. Laryng. Rhinol.*, **33**, 734.
—— (1955) *Z. Laryng. Rhinol.*, **34**, 271.
KUGELMASS, I. N. (1959) "Biochemistry of Blood". Oxford.
MAWSON, S. R. (1958) *J. Laryng.*, **72**, 56.
OBREGON, G. (1959) *A.M.A. Arch. Otolaryng.*, **70**, 297.
PAGET and CONWAY: *cited* by Guilford and Wright.
PERSILLON, A. (1957) *J. franç. Oto-rhino-laryng.*, **6**, 1.
PIETRANTONI, L., and BOCCA, E. (1958) *Ann. Otol.* (*St. Louis*), **66**, 1160.
DE PREST, R. A. (1954) *Acta Oto-rhino-laryng. belg.*, **8**, 691.
PRUVOT, M. (1956) *Rev. Laryng.* (*Bordeaux*), **77**, 261.
RAMBO, J. H. T. (1957) *A.M.A. Arch. Otolaryng.*, **66**, 525.
RAUCH, S. (1958) *Pract. oto-rhino-laryng.* (*Basel*), **20**, 361.
RICHTNÈR, N. G. (1958) *J. Laryng.*, **72**, 67.
WILLIAMS, H. L. (1958) *A.M.A. Arch. Otolaryng.*, **68**, 574.
WULLSTEIN, H. (1959) *J. Laryng.*, **73**, 515.
ZÖLLNER, F. (1954) *Acta oto-laryng.* (*Stockh.*), **44**, 370.
—— (1957) *Arch. Ohr.-Nas.- u. Kehlk.-Heilk.*, **171**, 1.

COMMENT ON THE STERILIZATION OF HANDPIECES FOR SURGICAL USE

By COLIN M. JOHNSTON (Swindon)

I WOULD like to congratulate Mr. M. Spencer Harrison and Mr. G. T. Hutchinson on their informative article on the power-driven bur in temporal bone surgery which will be of great value to all who use this now essential tool.

Faulty methods of sterilization of the handpiece are the most frequent cause of trouble which is usually due to rusting of the moving parts, resulting in seizure of the bearings and excessive wear from inadequate lubrication. With proper care, adjustment to take up wear in the bearings or the chuck are rarely necessary. Several years' experience of sterilization, using boiling A.C.10 emulsion has shown that seizure was not infrequent and the drill speed was reduced by friction resulting in heating of the handpiece when run continuously for several minutes. Furthermore, it is now generally considered that immersion in boiling water is not always adequate to ensure sterilization. The constant occurrence of rust-stained A.C.10 oil emulsion of doubtful sterility running down on to the drill point stimulated the writer to investigate a better and safer method of sterilization. For the last six months all handpieces used by the E.N.T. and Dental departments at the Princess Margaret Hospital, Swindon, have been sterilized in a hot air oven using a special heat resistant lubricating oil. To test the effectiveness of the sterilization a culture of *B. stearothermophilis* was inserted on a small piece of paper in the chuck mechanism in the centre of the handpiece and showed that the organism had been killed. Although the standard straight or contra-angled handpiece used for otological or mouth surgery is not harmed by this heat, the lubricating oil normally used becomes oxidized and loses its properties. Shell Tellus Oil*27, supplied by Messrs. Shell-Mex and B.P. Ltd.,* contains additives stated to inhibit oxidation and rusting, and to confer enhanced oiliness and anti-wear properties. It is stated to stand up to conditions of dry heat without oxidation. Experience has shown that these claims are justified and in particular the speed of rotation and smooth running of the drill have been entirely satisfactory. The handpiece reaches the surgeon free from moisture and there is no risk of oil seeping down the shaft of the drill.

The method of sterilization is as follows. The cover exposing the chuck

* P.O. Box No. 232. Shell B.P. House, Wine Street, Bristol, 1.

mechanism is removed and the bearings wiped with a cleaning cloth. If water has entered the handpiece, or in any case after it has been used six times, the handpiece is completely dismantled, including removal of the chuck ring from the tube. As the handpiece is re-assembled, a light smear of oil is applied to all working parts, over oiling being avoided. The handpiece is then placed in a tubular box such as is used for the sterilization of syringes and capped with thick metal foil. It is then placed in the oven at a temperature of 175° C. (347° F.) for 1½ hours. It is strongly advised that a dental colleague or a dental mechanic should instruct the technical staff responsible for the sterilization how the handpieces are constructed and assembled and that the mechanic should periodically make the necessary adjustments to take up wear.

I would like to differ from the authors in their preference for a flexible shaft drive from the motor to the handpiece when used with the standard motor giving 6,000 revolutions per minute. Even at full speed slight increase on the pressure of the cutting edge of the drill against the bone will cause an increased torque on the flexible shaft. It will then undergo longitudinal twisting like the elastic of a model aeroplane and then unwind itself, creating a disturbing lateral jump to the drill tip which may be displaced and cause damage to a neighbouring vital soft structure. The longer the shaft the more violent will be the recoil. A cord drive can be made considerably longer and allowed not only greater flexibility at the wrist without increase of friction but can be rested close at hand on the towels or instrument trolley without risk of swinging and touching non-sterile objects. For those who are ambidexterous in the use of the drill, the stand can be placed behind the operator and the horizontal arm swung over the surgeon's head from side to side as required. Jumping of the drill tip cannot occur as the cord will slip over the final pulley if excessive pressure is applied. With practice the surgeon can operate a foot control from the seated position and some may thus be able to stop the drill in case of sudden need more rapidly than by giving a verbal order. A spring loaded reversing foot control has the great additional advantage that on release, the foot lever swings beyond the "off" position and makes momentary contact with the first resistance stud driving the motor in the opposite direction. As a result the drill stops instantly without any over-run from inertia of the rotor of the electric motor.

CLINICAL RECORDS

HIDRADENOMA OF THE EXTERNAL AUDITORY MEATUS ASSOCIATED WITH FACIAL PALSY AND CHRONIC OTITIS MEDIA

By S. P. NANDI and H. J. SHAW (Croydon)

SWEAT gland tumours of the external auditory meatus are rare, especially in association with other chronic aural diseases. In the case presented here, chronic otitis media with ipsilateral facial palsy were synchronous features.

Differentiation and grading is often difficult and according to Azzopardi (1958) they may be regarded as a variant of the muco-epidermoid group of Stewart, Foote and Becker (1945).

Reports in the literature are uncommon: Fraser (1930) described two cases of "Malignant adenoma" of the external auditory meatus in two females, recurrent and causing the death of both patients, in one case 11 years after local excision and subsequent irradiation.

Johnstone, Lennox and Watson (1957) describe five cases seen in Glasgow from 1947-1955 and discuss their pathology in detail, finally deprecating the use of the term "ceruminoma". They consider them to be similar to cutaneous sweat gland tumours arising elsewhere, but their malignancy is difficult to predict and they have a greater tendency to local recurrence in the ear than at other sites.

All the three cases described by O'Neill and Parker (1957) also recurred despite energetic treatment and must be considered adenocarcinomata. One of the latter died with local recurrence and regional metastases to both sides of neck.

Kleinsasser and Scharfetter (1957) describe an unusual case of "ceruminal gland adenoma" invading the petrous temporal bone and posterior cranial fossa, apparently without any presentation in the external auditory meatus.

Juby (1957) gives an account of five other cases seen and treated in London at the Royal National Throat, Nose and Ear Hospital over the period of 1948-1956. Three of these were malignant, one having a facial nerve palsy on the same side.

Histologically these tumours should be regarded as adenoid neoplasms of the specialized sweat glands in the meatus. In a general sense they are often called "ceruminomata", but this is not a preferred term, since it is inaccurate from the standpoint of both pathology and descriptive precision. The cerumen derives from several sources, only one of which is the secretion of the sweat glands; moreover, the tumour is a glandular neoplasm. An acceptable alternative term to Hidradenoma might rather be a tumour of mixed salivary type.

Clinical Records

Case Report

The patient was a female aged 79 years, severely crippled with osteo-arthritis and already under treatment for heart failure. She was first seen in the E.N.T. Department of the Croydon Hospitals in *November, 1960*, complaining of severe right-sided earache, intermittent headache, attacks of dizziness and right-sided facial palsy for the previous 2 weeks. She gave a past history of intermittent right aural discharge and gradually increasing deafness on the same side over the last 16 years. In addition her own doctor stated that the right external auditory meatus had been narrowed for several years.

She appeared pale, overweight, apprehensive, able to walk only with sticks and breathless on mild exertion. In addition, a complete right-sided facial palsy of lower motor neurone type was present. Examination of the right ear revealed a meatus completely occluded by what appeared to be a total collapse of the roof and posterior meatal wall. It was impossible to introduce a speculum painlessly beyond the obstruction, which although soft, slightly tender and only mildly resistant, seemed to extend to the depths of the meatus. It was impossible to see the tympanic membrane, but a slight offensive mucoid discharge was present. There was no useful hearing on the right side, conversational voice being heard at contact only. Fortunately, the left ear was normal and no abnormality was found in her upper air passages or neck. X-rays of the temporal bones revealed sclerotic mastoid processes and no gross sign of cholesteatoma.

Nevertheless a diagnosis of chronic otitis media and mastoiditis with cholesteatoma was made and in view of the patient's age and poor general condition it was decided to explore the right mastoid under xylocaine anæsthesia with heavy premedication.

Premedication

Seconal 1·5 gr. 2 hours pre-operative.

Morphia 0·25 gr. and *Atropine* 0·01 gr. with *Chlorpromazine* 0·75 gr. 1 hour before operation.

Operation

After extensive infiltration with *xylocaine* 2 per cent. solution the right mastoid was approached through a post-aural incision. On exposing the bony posterior and superior walls of the meatus it was seen that they were intact but that a moderately large and well-defined soft tissue tumour was present entirely within the membranous posterior meatal wall, extending from the introitus almost up to the tympanic membrane. The mastoid process itself was sclerotic and the few cells remaining around the antrum contained granulations, pus and cholesteatoma. A moderately large cholesteatoma occupied the expanded antrum, aditus and middle ear. Although there was some superficial erosion of bone, no definite fistula of the lateral semicircular canal was seen. The tympanic membrane was completely destroyed and cholesteatoma debris with a small aural polypus appeared to be impacted in the depth of the meatus by the presence of the meatal tumour.

A radical mastoidectomy was carried out with clearance of all diseased bone

and middle-ear remnants. Excision of the meatal tumour was facilitated by an additional end-aural incision. The removal of the tumour and its adnexae prevented utilization of any meatal plastic flap. The cavity was packed with BIPP gauze and both incisions closed by simple suture.

FIG. 1.
Cut surface of the meatal tumour.

The patient stood the operation surprisingly well and declared at the end that she had felt no pain but that she was glad when the hammering stopped.

Pathology Report on the Meatal Tumour

"Mixed salivary type tumour arising from external auditory meatus." The specimen measured 3·5 cm. × 1·3 cm. and was covered with skin. The cut

surface showed an oval apparently encapsulated mass (1·7 cm. × 1·3 cm.) with uniform solid grey appearance. A very small epidermal cyst was present between the skin and the tumour.

Sections show a tumour of the mixed salivary type, in this situation probably of sweat gland origin. There was no true capsule and the stroma was in the greater part hyaline resembling cartilage matrix, in other areas it was loose and spongy. Throughout the stroma were numerous glands lined mainly by a single

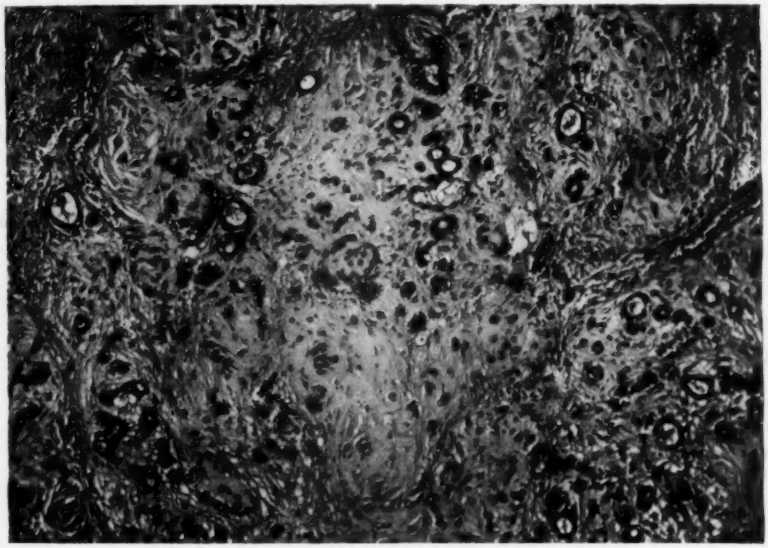

FIG. 2.

Hyaline stroma of the tumour interspersed with numerous glandular spaces and epithelial elements. × 75.

layer of cuboid epithelium. Similar epithelial cells formed solid trabeculae in the stroma and small single groups were scattered throughout.

The secretions in the glands reacted strongly to the P.A.S. method. Alcian blue did not stain the material but stained the few strands lying between this central secretion mass and the glandular epithelium. There was a heavy mottling of the acid mucopolysaccharide (Alcian blue positive) throughout the cartilaginous like stroma.

Although the ear healed well and improvement in the facial palsy was detected within the first post-operative week, her general condition delayed discharge from hospital for 3 weeks. By the fourth week the facial palsy had completely recovered and she was free from pain and dizziness. At her attendance as an outpatient 8 weeks after the operation the mastoid cavity was clean and almost dry.

Discussion

This case is reported principally owing to the rarity of hidradenomata of the external auditory meatus and because of the co-existing cholesteatoma with chronic otitis media. Prior to surgery the latter condition had been correctly diagnosed as the cause of her symptoms and facial palsy, but the presence of the meatal neoplasm was only revealed at operation. Therefore the case also emphasizes the constant need for awareness of a double pathology complicating the more obvious lesion. Moreover, although the tumour was apparently benign the facial palsy could certainly have been explained by the presence of an adenocarcinoma.

A final point of interest lay in the apparently complete effectiveness of the premedication and the 2 per cent. xylocaine infiltration anæsthesia, and in the comparatively mild shock of hammer and gouge. Contrary to most expectations, it has been said that the use of the burr produces far greater discomfort owing to noise and vibration.

Acknowledgements

The authors wish to thank Dr. D. J. O'Brien, Pathologist of the Croydon Hospitals Group, very sincerely for the photomicrograph and specimen report.

REFERENCES

AZZOPARDI, J. G. (1958) *J. Path. Bact.*, **76**, 379.
FRASER, J. S. (1930) *J. Laryng.*, **45**, 636.
JOHNSTONE, J. M., LENNOX, B., and WATSON, A. J. (1957) *J. Path. Bact.*, **73**, 421.
JUBY, H. B. (1957) *J. Laryng.*, **71**, 832.
KLEINSASSER, O., and SCHARFETTER, G. (1957) *Neurochir.*, **17**, 4.
O'NEILL, P. B., and PARKER, R. A. (1957) *J. Laryng.*, **71**, 824.
STEWART, F. W., FOOTE, F. W., and BECKER, W. F. (1945) *Ann. Surg.*, **122**, 820.

7 Wimpole Street,
London, W.1

A CASE OF PERICHONDITIS AND NECROSIS OF LARYNGEAL CARTILAGE 25 YEARS AFTER TREATMENT WITH RADIUM

By F. C. ROBSON and J. D. K. DAWES (Newcastle-upon-Tyne)

Patient E.P., male aged 75

In *September, 1934* this patient presented with a 9-month history of hoarseness. A diagnosis of squamous cell carcinoma of the right vocal cord was made

FIG. 1.

Anterior view of neck showing abscess over the thyroid cartilage.

and later confirmed by biopsy. A Finzi-Harmer operation was performed. 5 × 0·8 mg. radium needles were inserted and left in position for 10 days. At

no time in the immediate post-operative phase was there any sign of perichondritis. In 1945 it was noted that the right vocal cord had become fixed.

In *November, 1958*, this patient presented with a swelling in the front of the neck of 4 days duration.

Examination

A fluctuant swelling on the front of neck in the region of the old scar, which was fixed to the larynx. Full routine examination revealed no other physical signs.

A horizontal incision was made at the lower end of the swelling and the wound drained.

Bacteriology showed *Streptococcus viridans* sensitive to all antibiotics. The patient was put on to penicillin and streptomycin.

As the wound was still oozing pus after 5 days a further swab was taken for bacteriology which showed *Hæmophilus influenzae* sensitive to chloromycetin. The antibiotic was therefore changed. This did not improve the condition so it was decided to explore the abscess.

Operation

A trap door flap was lifted as there were already vertical and horizontal incisions present. There was obvious necrosis of cartilage at the level of the thyroid notch and a portion of the thyroid alar medial to the oblique line. The soft tissue showed some necrosis in the region of the right vocal cord, and there was a hole directly into the laryngeal airway. The wound was closed in layers with a drain. A keyhole tracheotomy was made below as a precautionary measure. The histology of the fragments removed showed fragments of bone and chronic inflammatory change. The wound healed slowly over the next 2 weeks, the antibiotic being changed back to penicillin after a further bacteriology report. The wound has remained closed.

Discussion

It is well known that radium necrosis may follow interstitial irradiation for a carcinoma of the vocal cord. On occasions although the larynx has remained well, an acute upper respiratory tract infection may precipitate an acute perichrondritis some years after the irradiation. However, it is unusual to see a perichondritis developing 25 years after radiotherapy, and we presume that irradiation reduced local resistance of the tissues and that an upper respiratory tract infection precipitated this perichondritis which continued to the state of gross cartilaginous necrosis.

Antibiotics and removal of the sequestrum were sufficient to produce resolution of the disease.

MÉNIÈRE'S DISEASE IN A DEAF MUTE

By W. DOYLE KELLY (Dublin)

MRS. A.L., aged 33, first attended the E.N.T. Out-patient Department of Sir Patrick Dun's Hospital on *14.8.58* complaining of noises in both ears since *November, 1957*, and an attack of rotatory vertigo accompanied by nausea and vomiting, which persisted for 3 hours on a single occasion. This took place about 1 week prior to her first visit. Tinnitus had been particularly troublesome for 2 days preceding the onset of the vertigo. The patient was accompanied by her mother, who stated that Mrs. A.L. had been deaf and dumb since birth, and this deafness was noticed at 2 years of age. At 6 months old the patient had pneumonia, but this was not accompanied by otitis media or any of the exanthemata. There was no family history of deafness, mental defect, syphilis or maternal rubella, and no personal history of birth trauma, jaundice or Rh. incompatibility. The patient's mother has had a successful craniotomy performed for what was presumably a ruptured cerebral aneurism 8 years ago. Mrs. A.L. attended an institution for the deaf and dumb between the ages of 7 and 17 years, where, at the time, sign language was the method of communication taught.

The patient herself possesses normal intelligence, but her attempt at speech is unintelligible, her principal method of communication, in addition to sign language in her immediate family, is by the written word and she writes an excellent hand with great rapidity. She has five normally hearing children, even though, the father of these children is himself a deaf mute since birth.

On examination the tympanic membranes and external auditory meati were quite normal. Audiometric signals were appreciated at 512 and 1,024 frequencies at the 100 decibel level. I hesitate to use the word "heard" as shouting *ad concham* did not appear to be perceived.

Caloric labyrinthine tests showed the following result (Fig. 1).
Pilocarpine grains $\frac{1}{8}$, and Phenobarbitone grains $\frac{1}{2}$ were given twice daily for 2 months. There was no further attack of vertigo in that time, so treatment was discontinued and a further caloric test done (Fig. 2).

Two weeks later the patient returned to say that further vertiginous attacks had taken place over 5 days and the last one had lasted for 4 hours. Pilocarpine and phenobarbitone treatment was re-started for a further 4 weeks with good result, and a period of remission was enjoyed until *11.3.59*. At this visit Mrs. A.L. complained that she had been dizzy all day for each of the previous 4 days. No nystagmus was demonstrated, but a caloric test gave the following result (Fig. 3).

It was then decided to perform membranous labyrinthectomy on the left ear. Whilst awaiting admission for operation the patient had become pregnant for the fifth time, and it was decided to postpone the labyrinthectomy. During preg-

nancy the patient had infrequent attacks of vertigo, especially during the latter 5 months. Attacks became more frequent in the puerperium and in May her caloric test showed the following reactions (Fig. 4).

X-rays demonstrated normally pneumatized mastoid air cell systems in each ear and normal middle-ears and internal auditory meati.

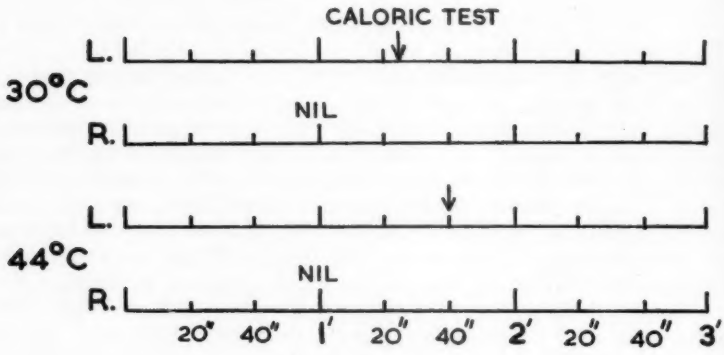

FIG. 1.

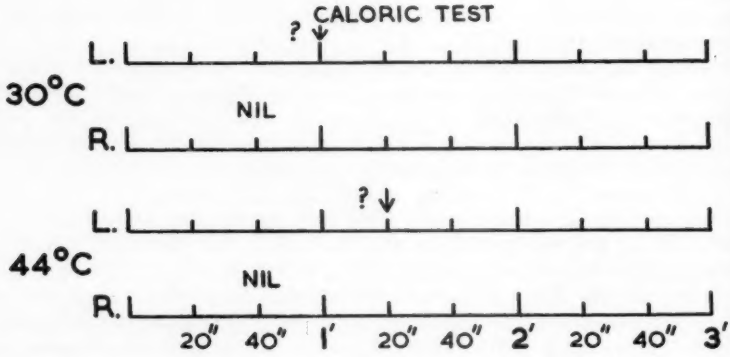

FIG. 2.

Operation

On *6.5.1960*, the left lateral semi-circular canal was exposed through an endaural incision and a normally cellular mastoid. The endolymphatic duct was normal in size and proportion as far as could be seen with the operating microscope. The duct was avulsed and a bone chip plug inserted into the fenestra. The wound was then closed. Penicillin and sulphonamide were given for the first post-operative week. The patient remained in hospital for 3 weeks as she had more post-operative vertigo than I would have thought possible judging

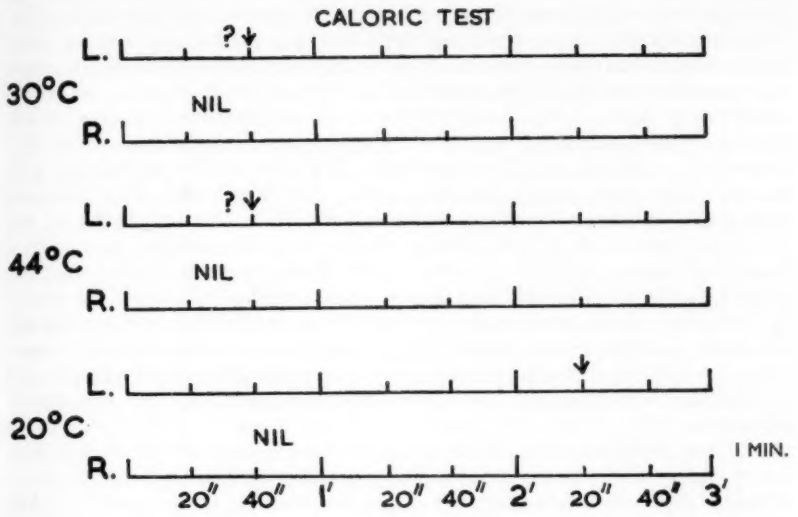
CALORIC TEST
L.
?
30°C
NIL
R.
L.
?
44°C
NIL
R.
L.
20°C
NIL
R.
1 MIN.
20″ 40″ 1′ 20″ 40″ 2′ 20″ 40″ 3′

FIG. 3.

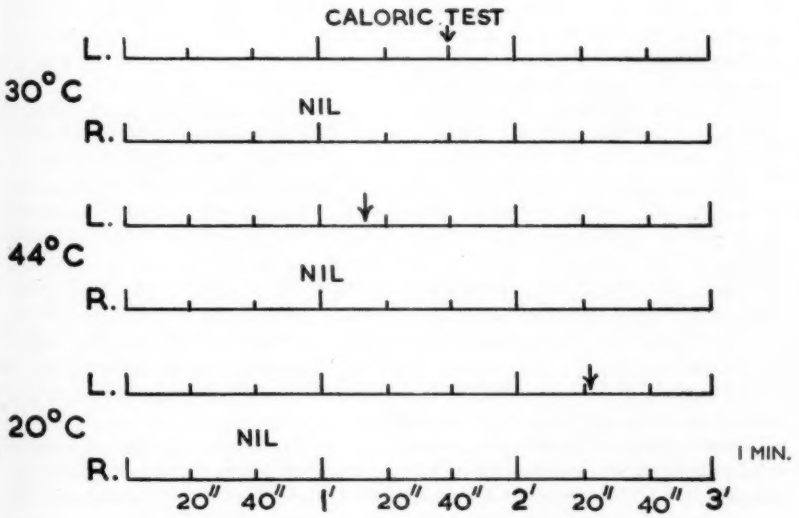
CALORIC TEST
L.
30°C
NIL
R.
L.
44°C
NIL
R.
L.
20°C
NIL
R.
1 MIN.
20″ 40″ 1′ 20″ 40″ 2′ 20″ 40″ 3′

FIG. 4.

by the pre-operative lack of response on caloric-testing the right labyrinth. Rehabilitation was advanced by post-operative exercises in the hands of the Physiotherapy Department. In the immediate post-operative period Mrs. A.L. showed horizontal nystagmus and complained of unsteadiness, noise in the ears and later difficulty in focusing objects while she was in motion and at one period stated that she "saw two of everything". The horizontal nystagmus settled down in 3 weeks and the oscillatory type was never in evidence. Gait was affected, particularly with the eyes closed. My note of *28.7.1960* reads "gait normal with eyes open, improving with eyes shut". On *14.11.1960* an audiogram revealed no hearing in the operated ear, but "hearing" at 95 db. for 512 and 1,024 on the right side. Tinnitus was less and the vision was described as "still a bit weak when walking in the dark". There was no induced nystagmus on stimulation of either side by a flow of water at 20° C. for one full minute.

When seen in *January, 1961*, the patient has stated that she has had no attack of vertigo or tinnitus since her operation, and that whatever post-operative difficulties she did experience have now completely disappeared.

A review of the literature reveals two reported cases of Ménière's disease in deaf mutes.

Walter Dandy's case is that of a man of twenty-two years of age who had never heard nor spoken. Incapacitating attacks of recurrent vertigo in a rotatory plane with nausea and vomiting occurred three months previous to his first visit to Dandy's Clinic and these were accompanied by a sense of fullness in the right ear. "The attacks were heralded by a noise in the right ear (although he had no hearing)." The quote, including the parenthesis is Dandy's.

An audiogram showed response at the following frequencies, right side, 90 and 100 db. at 512 and 1024: left side, 85 and 105 db. at 512, 1024 and 2048, and on these Dandy comments as follows "certain wave frequencies were consistently registered as 'noises' and the range of sounds was less on the affected side. That these waves were actual sound waves, though not appreciated as sound was shown by the fact that after total division of the right VIIIth nerve all sounds were lost on that side. There was no appreciation of any wave frequency".

In sectioning the VIIIth nerve through the posterior fossa in this case no internal auditory meatus was present, the nerve passing directly into the petrous bone was smaller and deeper than normal. The facial nerve was anterior, angulated and separated from the auditory nerve rather than being concealed by the latter. Total section of the acoustic nerve was made by electro-cautery. The patient made an uneventful recovery and left hospital 1 week later.

The congenital malformation described had not been seen by Dandy in any of his earlier 640 cases. The patient following operation had no disturbance of equilibrium and he concluded, I think incorrectly, that this demonstrated the presence of function on the opposite side.

Gooch and Hallberg report a second case of Ménière's disease in a deaf mute subject, a man of 23 years of age. He had been born deaf and dumb by normal delivery and at two years of age had scarlet fever without otitis media. In this case nausea, vomiting and dizziness had been present all his life, but were worse in the 4 years preceding his registration at the Mayo Clinic. The first attack of vertigo occurred at the age of 7, thereafter at infrequent intervals until the age of 19 when he left an institution for the deaf and dumb.

Symptoms ordinarily persisted for a day or two, but latterly for fully 2 weeks. In addition to vertigo he complained of bilateral tinnitus and diplopia on occasion. Medical treatment was tried, but it failed to control the condition. As symptoms seemed bilateral and as there was very little reaction in response to caloric tests of both labyrinths, bilateral labyrinthectomy by Day's technique through normal well pneumatized mastoid cells was performed. Platignum electrodes were passed through fenestrae into each labyrinth and diathermy applied, sufficient current being used to cause facial twitches, but no paralysis was noted later. Penicillin and sulphonamide were given post-operatively and the patient hospitalized for a further 11 days. Severe post-operative vertigo was reported but this diminished rapidly.

Nine months post-operatively the patient reported that he had experienced only mild occasional dizziness without nausea and that this was diminishing in intensity. He was working a full day and supporting both himself and his deaf mute wife. He had, however, some diplopia, brought on by walking, which was accompanied by a pendular type of nystagmus.

Gooch and Hallberg refer to Dandy's case as being clearly unilateral of symptoms and selection of the responsible ear therefore possible, and these authors were forced to the reluctant decision that both ears needed operation in their own case.

A number of points arise out of the description of these three reported cases of Ménière's disease in the deaf mute. Briefly these points may be stated under the following headings:

(1) Tinnitus.
(2) Nystagmus and vertigo.

Tinnitus

The fact that a deaf mute should complain of tinnitus need occasion no surprise as it has been demonstrated that islands of sound perception beyond the limits of practical utilization exist. The presence of "islands of sound" prior to, and their disappearance, following on destruction of the end-organ or the severance of its connections with the sensorium would seem to indicate that a means of appreciating tinnitus does exist.

Two of the three cases may well belong to the Scheibe's type of deaf mutism in which sacculo-cochlear malformation is present. In this type the utricle and canals are intact and hearing remnants may be present. According to J. S. Fraser 70 per cent. of cases of deaf mutism belong to this category.

Vertigo and Nystagmus

Following on surgical destruction of the labyrinth vertigo accompanies nystagmus and both vertigo and nystagmus are in the horizontal plane after removal of the endolymphatic duct from the lateral semi-circular canal. This is often surprisingly active in spite of marked depression of the caloric reaction estimated pre-operatively in cases of Ménière's disease, but in general, this sign and symptom disappear in the course of 2 to 3 weeks.

Gooch and Hallberg's patient who had both labyrinths diathermized experienced post-operative dizziness. "This difficulty was characterized by an unusal

type of nystagmus and a tendency to over-shoot when the patient turned the eyes from any position back to directly forward. This continued briefly, in pendulum like fashion, until the eyes came to rest on the fixation object."

This type of vertigo and nystagmus is less usual certainly than the horizontal type and is seen in the following instances:

(*a*) Occasionally, following removal of packing over the meatal flap after fenestration of the lateral canal for otosclerosis, a pendular nystagmus may be observed for some days, and this is thought to be due to pulsating œdema in the flap, communicating impulses directly to the labyrinth.

(*b*) Directly arising from postural tests in occasional cases of paroxysmal positional vertigo.

(*c*) As a symptom of streptomycin toxicity.

The author had occasion to observe some 300 cases of genito-urinary tuberculosis, many of whom displayed varying degrees of damage to the cochlea and labyrinth. The majority of these patients had nephrectomy and were being treated for active infection in the remaining kidney. Dosage of streptomycin, was, at that time, relatively large, in the order of one to two grammes per day. Of those who complained of vertigo a very small proportion gave a history of inability to fixate an object when either they, or the object, were in motion. Fixation became sharp when the object became steady, or when the patients movement ceased. In these cases I failed to demonstrate any pendular movement of the eyes.

REFERENCES

DANDY, W. E. (1945) *Arch. Surg. (Chicago)*, **50**, 74.
FRASER, J. S. (1932) *Proc. roy. Soc. Med.*, **25**, 861.
GOLDSTEIN, M. A. (1939) The Acoustic Method for the training of the Deaf and Hard of Hearing Child. St. Louis.
GOOCH, J. O. and HALLBERG, O. E. (1947) *Proc. Mayo Clin.*, **22**, 252.

3 Fitzwilliam Place,
Dublin

GENERAL NOTES

TEMPLE UNIVERSITY SCHOOL OF MEDICINE

PHILADELPHIA 40. PA.

A "Short course in Audiology" is offered several times each year by the Section of Audiology, Department of Otorhinology, Temple University School of Medicine, Philadelphia 40, Pa.

The course will be one week in length and will encompass theoretical and clinical aspects of audition. Hearing testing and interpretation will be stressed. Office procedures, equipment and Industrial Audiometry will be discussed.

Further information may be obtained by writing to Philip E. Rosenberg, Ph.D., Director, Section of Audiology, Temple University Medical Center, 3401 North Broad St., Philadelphia, Pa.

SIXTH INTERNATIONAL CONGRESS OF AUDIOLOGY

THE sixth International Congress of Audiology will be held in Leyden, The Netherlands, from September 5th to 8th, 1962.

President: Prof. Dr. H. A. E. van Dishoeck;

Secretary Drs. A. Spoor.

In the programme are planned three round-table talks, with associated and free papers.

The subject of the round tables are:

"Frequency analysis of the normal and pathological ear."
Moderator: Prof. Dr. G. von Békésy.
"Central deafness in children."
Moderator: Prof. Dr. J. M. Tato.
"Psychogenic deafness and simulation."
Moderator: Prof. Dr. H. A. E. van Dishoeck.

Official languages of the Congress are: English, French, German and Spanish.
Working languages will be: English and French.

The address of the secretariat is:

Ear-Nose-Throat Department,
Academisch Ziekenhuis,
LEIDEN (The Netherlands).

General Notes

NOTICE

An intensive postgraduate course in stapes surgery will be given May 14th to June 1st, 1962, by Dr. Samuel Rosen and Staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University.

The course will include all surgical techniques and the audiological principles involved. Surgery in the operating room and cadaver material will be used for instruction.

Candidates for the course must have had adequate experience in endaural surgery and must have available 50 cadavers for practice after termination of the course before operating upon patients.

Apply to *Registrar for Postgraduate Medical Instruction*, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, New York.

BACK JOURNALS REQUIRED AND FOR SALE

If any subscriber can spare copies of the January 1945 and June 1950 issues of the *Journal of Laryngology & Otology* they would be very much appreciated by the Librarian of The National Institute for the Deaf, 105 Gower Street, London, W.C.1.

If anyone would like to buy a set of the Journals from 1921 to 1960 (mostly bound volumes), and the two relevant indexes, etc., please apply to Donald G. P. Macalister, 5 Clarendon Place, Leamington Spa.

THE MOUNT SINAI HOSPITAL

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given January 13th, 1962 to January 26th, 1962 by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York in affiliation with Columbia University.

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, N.Y.

UNIVERSITY OF BAGHDAD

The University of Baghdad has asked the British Council to find British candidates for a number of posts in their Colleges of Medicine at Mosul and Baghdad. One of these appointments is for a Professor of Ear, Nose and Throat Diseases at the College of Medicine, Baghdad.

Anyone interested should communicate with the Director, Recruitment Department, The British Council, 65, Davies Street, London, W.1.

General Notes

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY

"THE GEORGE SEED MEMORIAL PRIZE" (previously known as the "Fifty Guinea Prize") will be awarded in 1962 for AN ORIGINAL PAPER on work in Otolaryngology carried out in the NORTH Of ENGLAND. The investigation must have been carried out during the tenure of a post as senior registrar, house officer, registrar or clinical assistant.

Entries (3 copies) should be sent, before June 30th, 1962, to:

The Honorary Secretary, North of England Otolaryngological Society,
73, Rodney Street, LIVERPOOL, 1.

THE INSTITUTE OF LARYNGOLOGY AND OTOLOGY

330-332, GRAY'S INN ROAD, LONDON, W.C.1.

associated with

THE ROYAL NATIONAL THROAT, NOSE & EAR HOSPITAL

NON-MEDICAL COURSES

November 6th, 1961 to *February 2nd, 1962*—Training course for audiology technicians.

May 30th and 31st and *June 1st, 1962*—Hearing and mental assessment of the deaf child—for psychologists.

The number of entries is limited in each course. A detailed syllabus of any of the above can be obtained on application from the Secretary-Administrator.

SHORT COURSES

for

CONSULTANTS AND SENIOR REGISTRARS

March 6th, 7th and 8th, 1962—Diagnosis and treatment of the deaf child.

April 27th and 28th, 1962—Practical Aural Surgery Class.

General Notes

MEETINGS & SEMINARS

especially arranged for

GENERAL PRACTITIONERS

who are cordially invited to attend all or any of the following without fee. Tea is available in the Refectory from 4.30 p.m. onwards on Fridays.

Sunday, December 3rd, 1961—10.15 a.m. "Seminar—Common ear, nose and throat conditions."—MR. WALLACE BLACK.

Friday, December 8th, 1961—3.30 p.m. "Pulmonary disease in relation to E.N.T." —DR. NORMAN HULBERT.

Friday, January 5th, 1962—3.30 p.m. "Treatment of acute inflammation in the ear."—MR. WILLIAM MCKENZIE.

Friday, January 19th, 1962—3.30 p.m. "Vasomotor and allergic rhinitis—chronic rhinitis."—MR. WALLACE BLACK.

Friday, February 2nd, 1962—3.30 p.m. "Disorders of blood in E.N.T. practice." —DR. D. A. OSBORN.

Friday, February 16th, 1962—3.30 p.m. "Vertigo and its differential diagnosis." —DR. C. H. EDWARDS.

Sunday, February 25th, 1962—10.15 p.m. "Seminar—Common ear, nose and throat conditions."—MR. WILLIAM MCKENZIE.

Friday, March 2nd, 1962—3.30 p.m. "Malignant diseases of the larynx—hoarseness."—MR. MAXWELL ELLIS.

Friday, March 16th, 1962—3.30 p.m. "Sinusitis."—MR. W. D. DOEY.

Friday, April 6th, 1962—3.30 p.m. "Dysphagia."—MR. G. H. HOWELLS.

Sunday, April 15th, 1962—10.15 a.m. "Seminar—Common ear, nose and throat conditions."—MR. W. D. DOEY.

Friday, April 27th, 1962—3.30 p.m. "Emergencies in throat, nose and ear practice."—MR. S. E. BIRDSALL.

Friday, May 4th, 1962—3.30 p.m. "Surgical treatment of deafness."—MR. K. MCLAY.

Friday, May 18th, 1962—3.30 p.m. "Chemotherapy in oto-laryngology."—DR. I. FRIEDMANN.

There is a limited number of appointments in the associated Hospital as Clinical Assistants and Out-Patient Assistants for which general practitioners are eligible to apply. Particulars of these can be obtained from the House Governor of the Hospital upon request.

General Notes

SPECIAL LECTURES AT THE INSTITUTE

February 3rd, 1962—12.0 noon. "Temporal bone pathology."—PROF. H. K. KRISTENSEN (Copenhagen).

March 15th, 1962—5.30 p.m. "On the pathogenesis of otosclerosis (hystochemical researches)."—PROF. M. ARSLAN (Padua).

May 5th, 1962—12.0 noon. "The clinical significance of malignant tumour cells in the blood."—PROF. A. HERRMANN (Munich).

ROYAL COLLEGE OF SURGEONS
OTOLARYNGOLOGY LECTURES

THE following lectures, arranged jointly by the Royal College of Surgeons of England and the Institute of Laryngology and Otology, will be delivered in the Lecture Hall of the College on Thursday afternoons at 5.30 p.m.

1962

February 1st. MR. GEOFFREY H. BATEMAN. "Hypophysectomy."

March 1st. MR. ALAN H. HUNT. "Tumours of the Upper Jaw."

May 3rd. DR. I. FRIEDMANN. "Electronmicroscopy in Otolaryngology."

FIRST INTERNATIONAL SYMPOSIUM
of the
MANHATTAN EYE, EAR AND THROAT HOSPITAL
PLASTIC AND RECONSTRUCTIVE SURGERY OF THE EYE, EYELIDS & ADNEXA

May 21st-25th, 1962

New York City

Honorary President: Prof. Varaztad H. Kazanjian.
Chairman: Prof. Richard C. Troutman.
Programme Chairmen: Prof. John M. Converse and Dr. Byron Smith.
Executive Committee: Prof. Joaquin Barraquer, Prof. Benjamin F. Boyd, Dr. Blair O. Rogers, Sir Benjamin Rycroft, Prof. Dr. Karl Schuchardt.
Registration fee: $100 (U.S.A. and Canada).
$ 50 (other countries).
For further information, please write to: Prof. Richard C. Troutman, Chairman, First International Symposium of the Manhattan Eye, Ear and Throat Hospital, 210 East 64th Street, New York 21, N.Y.

General Notes

FIRST BRITISH ACADEMIC CONFERENCE IN OTOLARYNGOLOGY

This will be held at the Royal College of Surgeons of England between June 16th and 21st, 1963.

There will be formal sessions at which invited speakers will read papers, and these will be discussed.

A special feature will be seminars and revision courses conducted for small groups by known authorities on various topics in our specialty and allied subjects. These will include the basic sciences.

Information about the Conference will be sent in due course to Otolaryngological Associations and Societies overseas, and members of these will be welcome as participants.

Further notifications will appear in this Journal from time to time.

OGY
etween
rs, and
r small
allied
ryngo-
will be